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     (FILE 'REGISTRY' ENTERED AT 07:53:14 ON 08 AUG 2000)
                DEL HIS
                ACT CRANE363A/A
               ---------
L1              1 SEA FILE=REGISTRY ABB=ON  PLU=ON  58-96-8
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                ACT CRANE363B/A
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L2   (         16)SEA FILE=REGISTRY ABB=ON  PLU=ON  (563-24-6/BI OR 5909-45-5/BI
L3   (          1)SEA FILE=REGISTRY ABB=ON  PLU=ON  58-96-8
L4   (          1)SEA FILE=REGISTRY ABB=ON  PLU=ON  9030-22-2
L5   (         14)SEA FILE=REGISTRY ABB=ON  PLU=ON  L2 NOT (L3 OR L4)
L6   (          4)SEA FILE=REGISTRY ABB=ON  PLU=ON  62-49-7 OR 67-48-1 OR 87-67-2
L7   (          2)SEA FILE=REGISTRY ABB=ON  PLU=ON  563-24-6 OR 987-78-0
L8   (          3)SEA FILE=REGISTRY ABB=ON  PLU=ON  35898-87-4 OR 54-03-5 OR 5983
L9   (          5)SEA FILE=REGISTRY ABB=ON  PLU=ON  L5 NOT (L6 OR L7 OR L8)
L10  (          4)SEA FILE=REGISTRY ABB=ON  PLU=ON  L9 NOT C9H11NO3
L11  (          3)SEA FILE=REGISTRY ABB=ON  PLU=ON  L10 NOT 65-46-3
L12             12 SEA FILE=REGISTRY ABB=ON  PLU=ON  (L6 OR L7 OR L8 OR L11)
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     FILE 'EMBASE' ENTERED AT 07:54:40 ON 08 AUG 2000
L13           2023 S L1
L14           2457 S URIDINE/CT OR URIDINE DERIVATIVE/CT
L15           2457 S L13,L14
L16          19386 S URIDINE DERIVATIVE+NT/CT
L17          16929 S L16 NOT L15
L18           8059 S L12
L19           7914 S (CHOLINE OR CHOLINE DERIVATIVE OR CITICOLINE OR DILAZEP OR HE
L20          16011 S (PHOSPHATIDYLCHOLINE OR LYSOPHOSPHATIDYLCHOLINE OR GLYCEROPHO
L21              0 S BENZYLBARBITURATE OR BENZYL BARBITURATE
L22              0 S BENZENE BARBITURATE
L23         147653 S FATTY ACID+NT/CT
L24           7265 S LECITHIN OR LYSOLECITHIN
L25            803 S CDP CHOLINE OR ACYLGLYCEROPHOSPHOCHOLINE OR ACYL GLYCEROPHOSP
L26             42 S CYTIDINE DIPHOSPHOCHOLINE
L27              1 S CYTIDINE DIPHOSPHO CHOLINE
L28             96 S L15 AND L18-L27
L29            108 S L15 AND (NEUROLOGIC DISEASE+NT OR MEMORY+NT OR NIEMANN PICK D
L30              2 S L15 AND (ATTENTION+NT OR ATTENTION DEFICIT DISORDER OR SELECT
L31             26 S L15 AND (BEHAVIOR+NT OR BEHAVIOR DISORDER+NT OR EMOTION+NT OR
L32             21 S L15 AND (PANIC OR ANXIETY OR ANXIETY NEUROSIS+NT OR TRANQUILI
L33             37 S L15 AND (TARDIVE DYSKINESIA OR THROMBOSIS+NT OR HYPOXIA+NT OR
L34            138 S L15 AND (BRAIN+NT OR BRAIN ATROPHY OR BRAIN INJURY+NT OR BRAI
L35             14 S L15 AND (SPINAL CORD+NT OR SPINAL CORD INJURY+NT OR SPINAL CO
L36              7 S L15 AND (POSTPOLIOMYELITIS SYNDROME OR POLIOMYELITIS OR POLIO
L37              5 S L15 AND (NEUROMUSCULAR FUNCTION+NT OR NEUROMUSCULAR DISEASE+N
L38            225 S L15 AND (NERVOUS TISSUE+NT OR NERVOUS SYSTEM+NT OR NERVOUS SY
L39            298 S L29-L38
L40            198 S L14/MAJ AND L39
L41             13 S L28 AND L40
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L42          183 S L15 AND A8./CT
L43           77 S L15 AND (F1. OR F2. OR F3. OR F4.)/CT
L44          162 S L15 AND (G2.600. OR C2.610. OR C6.450.610.)/CT
L45           31 S L15 AND A10.600./CT
L46          310 S L42-L45,L39
L47          205 S L46 AND L14/MAJ
L48         1123 S L15 AND (E2.230. OR G1.680. OR E5.20.240.)/CT
L49          359 S L14(L)(CT OR AD OR CB OR CM OR CR OR DV OR DO OR DT OR PR OR
L50         3300 S L16(L)(CT OR AD OR CB OR CM OR CR OR DV OR DO OR DT OR PR OR
L51          271 S L49 AND L14/MAJ
L52           38 S L51 AND L39,L46
L53           38 S L52 AND L48
L54           38 S L52,L53
L55            2 S L28 AND L54
L56          258 S L51 AND PY<=1998
L57           36 S L54 AND L56
L58           13 S L57 NOT AB/FA
L59           23 S L57 NOT L58

     FILE 'EMBASE' ENTERED AT 08:33:58 ON 08 AUG 2000

=> d all tot 158


L58  ANSWER 1 OF 13  EMBASE  COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN   97271841  EMBASE
DN   1997271841
TI   [How to get a grip on alcohol neuropathies].
     WIE DIE ALKOHOL-NEUROPATHIE IN DEN GRIFF ZU KRIEGEN IST.
AU   Filip K.B.
SO   Zeitschrift fur Allgemeinmedizin, (1997) 73/9 (527).
     ISSN: 0341-9835  CODEN: ZALMAS
CY   Germany
DT   Journal; Conference Article
FS   008     Neurology and Neurosurgery
     037     Drug Literature Index
LA   German
CT   Medical Descriptors:
     *alcoholism
     *polyneuropathy: DT, drug therapy
     conference paper
     drug efficacy
     human
     Drug Descriptors:
     *benfotiamine: DT, drug therapy
     *cytidine: DT, drug therapy
     *pyridoxine: DT, drug therapy
     *thiamine: DT, drug therapy
     *uridine: DT, drug therapy
     keltican
     neurotrat
     vitamin b complex
     unclassified drug
RN   (benfotiamine) 22457-89-2; (cytidine) 65-46-3; (pyridoxine) 12001-77-3,
     58-56-0, 65-23-6, 8059-24-3; (thiamine) 59-43-8, 67-03-8; (uridine)
     58-96-8; (neurotrat) 55015-09-3
CN   Neurotrat; Milgamma; Keltican

L58  ANSWER 2 OF 13  EMBASE  COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN   96238329  EMBASE
DN   1996238329
TI   [Polyneuropathies: New study on drug therapy of alcoholism].
     POLYNEUROPATHIEN: NEUE STUDIE ZUR AM-THERAPIE.
AU   Dietz G.
SO   Pharmazeutische Zeitung, (1996) 141/32 (53).
     ISSN: 0031-7136  CODEN: PZSED5
```

CY Germany
DT Journal; Note
FS 008 Neurology and Neurosurgery
040 Drug Dependence, Alcohol Abuse and Alcoholism
030 Pharmacology
037 Drug Literature Index
LA German
SL German
CT Medical Descriptors:
***alcoholism: DT, drug therapy**
***polyneuropathy: DT, drug therapy**
clinical trial
human
major clinical study
note
Drug Descriptors:
***benfotiamine: DT, drug therapy**
***cytidine: DT, drug therapy**
***nucleotide: DT, drug therapy**
***pyridoxine: DT, drug therapy**
***thiamine: DT, drug therapy**
***uridine: DT, drug therapy**
RN (benfotiamine) 22457-89-2; (cytidine) 65-46-3; (pyridoxine) 12001-77-3, 58-56-0, 65-23-6, 8059-24-3; (thiamine) 59-43-8, 67-03-8; (uridine) **58-96-8**

L58 ANSWER 3 OF 13 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 93024387 EMBASE
DN 1993024387
TI [Nucleotides against low back pain].
NUKLEOTIDE GEGEN KREUZSCHMERZEN.
SO TW Neurologie Psychiatrie, (1992) 6/12 (797).
ISSN: 0935-3224 CODEN: TWNPE3
CY Germany
DT Journal; Note
FS 008 Neurology and Neurosurgery
024 Anesthesiology
037 Drug Literature Index
LA German
CT Medical Descriptors:
***low back pain: DT, drug therapy**
***myopathy: DT, drug therapy**
***polyneuropathy: DT, drug therapy**
drug mixture
human
note
Drug Descriptors:
***cytidine: DT, drug therapy**
***cytidine: CB, drug combination**
***nucleotide: DT, drug therapy**
***uridine: DT, drug therapy**
***uridine: CB, drug combination**
keltican: DT, drug therapy
unclassified drug
RN (cytidine) 65-46-3; (uridine) **58-96-8**
CN Keltican

L58 ANSWER 4 OF 13 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 92246061 EMBASE
DN 1992246061
TI Treatment of diabetic polyneuropathy.
AU Jakobsen J.
CS Odense University Hospital, Odense, Denmark
SO Acta Neurologica Scandinavica, (1992) 86/1 (1-2).
ISSN: 0001-6314 CODEN: ANRSAS
CY Denmark

DT Journal; Editorial
FS 008 Neurology and Neurosurgery
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
CT Medical Descriptors:
*diabetes mellitus: TH, therapy
***diabetes mellitus: DT, drug therapy**
***diabetic neuropathy: ET, etiology**
***diabetic neuropathy: DT, drug therapy**
editorial
human
liver toxicity: SI, side effect
nonhuman
oral drug administration
priority journal
rash: SI, side effect
Drug Descriptors:
***aldehyde reductase: DT, drug therapy**
***insulin: DT, drug therapy**
***ponalrestat: DT, drug therapy**
***sorbinil: DT, drug therapy**
*sorbinil: AE, adverse drug reaction
***tolrestat: DT, drug therapy**
***uridine: DT, drug therapy**
RN (aldehyde reductase) 58591-34-7, 9023-11-4, 9028-31-3; (insulin) 9004-10-8; (ponalrestat) 72702-95-5; (sorbinil) 68367-52-2; (tolrestat) 82964-04-3; (uridine) **58-96-8**

L58 ANSWER 5 OF 13 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 91232207 EMBASE
DN 1991232207
TI Synthesis of brain-targeted 5-iodo, 5-vinyl- and (E)-5-(2-iodovinyl)-2'-deoxyuridines coupled to a dihydropyridine .dblarw. pyridinium salt redox chemical delivery system.
AU Kumar R.; Ji G.; Wiebe L.I.; Knaus E.E.
CS Faculty of Pharmacy and Pharmaceutical Sciences, University of Alberta, Edmonton, Alta. T6G 2N8, Canada
SO Journal of Heterocyclic Chemistry, (1991) 28/3 (711-715).
ISSN: 0022-152X CODEN: JHTCAD
CY United States
DT Journal; Article
FS 037 Drug Literature Index
LA English
CT Medical Descriptors:
*drug synthesis
article
blood brain barrier
drug targeting
Drug Descriptors:
*antivirus agent: AN, drug analysis
*antivirus agent: DV, drug development
***uridine derivative: AN, drug analysis**
***uridine derivative: DV, drug development**

L58 ANSWER 6 OF 13 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 91130550 EMBASE
DN 1991130550
TI The treatment of diabetic foot.
AU Schaper C.
CS Ermelestrasse 6, 7450 Hechingen, Germany
SO Zeitschrift fur Allgemeinmedizin, (1991) 67/6 (346).
ISSN: 0341-9835 CODEN: ZALMAS
CY Germany
DT Journal; Note

FS 003 Endocrinology
006 Internal Medicine
037 Drug Literature Index
LA German
CT Medical Descriptors:
*diabetes mellitus
***diabetic neuropathy: ET, etiology**
***diabetic neuropathy: DT, drug therapy**
*foot
human
note
prophylaxis
Drug Descriptors:
***cobalamin: DT, drug therapy**
***cytidine: DT, drug therapy**
***prostaglandin e1: DT, drug therapy**
***uridine: DT, drug therapy**
benfotiamine
prostavasin
RN (cobalamin) 13408-78-1; (cytidine) 65-46-3; (prostaglandin e1) 745-65-3; (uridine) **58-96-8**; (benfotiamine) 22457-89-2; (prostavasin) 55648-20-9
CN Prostavasin; Benfotiamine

L58 ANSWER 7 OF 13 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 91113095 EMBASE
DN 1991113095
TI Pharmacological analysis of uridine anxiolytic activity.
AU Karkishchenko N.N.; Khaitin M.I.; Simkina Yu. N.
CS Laboratory of Molecular Pharmacology, Research Institute of Physical and Organic Chemistry, State University, 194/3, prospekt Stachki, Rostov-na-Donu 344104, Russia
SO Farmakologiya i Toksikologiya, (1991) 54/1 (16-18).
ISSN: 0014-8318 CODEN: FATOAO
CY Russia
DT Journal; Article
FS 030 Pharmacology
037 Drug Literature Index
LA Russian
SL English
CT Medical Descriptors:
***tranquilizing activity**
animal experiment
article
mouse
nonhuman
serotoninergic system
Drug Descriptors:
***cyproheptadine: PD, pharmacology**
***haloperidol: PD, pharmacology**
***prazosin: PD, pharmacology**
***propranolol: PD, pharmacology**
***uridine: PD, pharmacology**
RN (cyproheptadine) 129-03-3, 969-33-5; (haloperidol) 52-86-8; (prazosin) 19216-56-9, 19237-84-4; (propranolol) 13013-17-7, 318-98-9, 3506-09-0, 4199-09-1, 525-66-6; (uridine) **58-96-8**

L58 ANSWER 8 OF 13 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 90300713 EMBASE
DN 1990300713
TI Chronic but not acute uridine treatment reduces haloperidol-induced dopamine release in neostriatum as studied by intracerebral microdialysis.
AU Ruggeri M.; Zoli M.; Ungerstedt U.; Peruzzi G.; Agnati L.F.; Fuxe K.
CS Institute of Human Physiology, University of Modena, Italy
SO Neuroscience Research Communications, (1990) 6/3 (187-192).
ISSN: 0893-6609 CODEN: NRCOEE

CY United Kingdom
DT Journal; Article
FS 037 Drug Literature Index
002 Physiology
LA English
CT Medical Descriptors:
***corpus striatum**
***dopamine release**
*microdialysis
rat
animal experiment
nonhuman
male
intraperitoneal drug administration
article
Drug Descriptors:
*dihydroxyphenylacetic acid
***haloperidol: CB, drug combination**
***uridine: PD, pharmacology**
***uridine: CB, drug combination**
RN (haloperidol) 52-86-8; (uridine) **58-96-8**
CN (1) Serenase
CO (1) Istituto lusofarmaco (Italy)

L58 ANSWER 9 OF 13 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 90294377 EMBASE
DN 1990294377
TI Hypnotic and sedative activities of N3-substituted uridine, thymidine and azauridine, and their action mechanism.
AU Yamamoto I.; Watanabe K.; Koshigami M.; Furuta E.; Tateoka Y.; Kimura T.; Ho I.K.
CS Department of Hygienic Chemistry, Faculty of Pharmaceutical Sciences, Hokuriku University, 3-Ho Kanagawa-machi, Kanazawa 920-11, Japan
SO European Journal of Pharmacology, (1990) 183/4 (1559-1560).
ISSN: 0014-2999 CODEN: EJPHAZ
CY Netherlands
DT Journal; Conference Article
FS 008 Neurology and Neurosurgery
030 Pharmacology
037 Drug Literature Index
LA English
CT Medical Descriptors:
***hypnosis**
***motor activity**
*sedation
brain homogenate
drug metabolism
mouse
structure activity relation
animal experiment
animal cell
nonhuman
intracerebroventricular drug administration
conference paper
priority journal
Drug Descriptors:
4 aminobutyric acid receptor
benzodiazepine receptor
radioisotope
***azauridine derivative: PD, pharmacology**
*azauridine derivative: DV, drug development
*azauridine derivative: DO, drug dose
***thymidine derivative: PD, pharmacology**
*thymidine derivative: DV, drug development
*thymidine derivative: DO, drug dose
***uridine derivative: PD, pharmacology**

***uridine derivative: DV, drug development**
***uridine derivative: DO, drug dose**

L58 ANSWER 10 OF 13 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 90107322 EMBASE
DN 1990107322
TI [Effects of chronic uridine treatment on the homeostasys of the dopaminergic synapse: Possible rilevance for therapy of schizophrenia]. EFFETTI DEL TRATTAMENTO CRONICO CON URIDINA SULL'OMEOSTASI DELLA SINAPSI DOPAMINERGICA: POSSIBILI IMPLICAZIONI PER LA TERAPIA DELLE SINDROMI SCHIZOFRENICHE.
AU Ruggeri M.
SO Neurologia Psichiatria Scienze Umane, (1989) 9/4 (529-544).
CODEN: NPSUEU
CY Italy
DT Journal; Article
FS 006 Internal Medicine
008 Neurology and Neurosurgery
032 Psychiatry
037 Drug Literature Index
LA Italian
SL English
CT Medical Descriptors:
***behavior**
aged
cytochemistry
rat
psychological aspect
animal cell
nonhuman
article
Drug Descriptors:
*cholecystokinin
***dopamine receptor**
***haloperidol: PD, pharmacology**
***haloperidol: DT, drug therapy**
***haloperidol: CB, drug combination**
***uridine: PD, pharmacology**
***uridine: DT, drug therapy**
***uridine: CB, drug combination**
RN (cholecystokinin) 9011-97-6, 93443-27-7; (haloperidol) 52-86-8; (uridine) **58-96-8**

L58 ANSWER 11 OF 13 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 90095511 EMBASE
DN 1990095511
TI [A study of the P300 and cerebral maps in subjects with multiinfarct dementia treated with the combination cytidine, uridine and l-glutamine]. DEMENZA MULTIINFARTUALE. STUDIO DELLA P300 E DELLE MAPPE CEREBRALI IN SOGGETTI CON DEMENZA MULTIINFARTUALE (MID) TRATTATI CON ASSOCIAZIONE CITIDINA, URIDINA ED L-GLUTAMINA.
AU Firenze C.; Mazzotta G.; Montesi S.; Gallai V.
CS Cattedra di Terapia Neurologica, Clinica delle Malattie, Nervose e Mentali, Universita degli Studi - Perugia, Perugia, Italy
SO Basi Razionali della Terapia, (1989) 19/11 (683-388).
ISSN: 0393-7569 CODEN: BRDPEQ
CY Italy
DT Journal; Article
FS 008 Neurology and Neurosurgery
030 Pharmacology
037 Drug Literature Index
LA Italian
CT Medical Descriptors:
***cerebrovascular accident**
***dementia: DT, drug therapy**
***evoked response**

adult
aged
human
psychological aspect
oral drug administration
article
Drug Descriptors:
***cytidine: PD, pharmacology**
***cytidine: DT, drug therapy**
***glutamine: PD, pharmacology**
***glutamine: DT, drug therapy**
***uridine: PD, pharmacology**
***uridine: DT, drug therapy**
RN (cytidine) 65-46-3; (glutamine) 56-85-9, 6899-04-3; (uridine) **58-96-8**

L58 ANSWER 12 OF 13 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 90066245 EMBASE
DN 1990066245
TI Ultrastructural alterations of different formations of the brain induced by uridin.
AU Karkishchenko N.N.; Bardakhchyan E.A.
CS Kafedra Klinicheskoj Farmakologii Fakul'teta Usovershenstvovanija Vrachej II Moskovskogo Meditsinskogo Instituta, Moskva, Russia
SO Byulleten Eksperimentalnoi Biologii i Meditsiny, (1990) 108/1 (86-89).
ISSN: 0365-9615 CODEN: BEBMAE
CY Russia
DT Journal; Article
FS 030 Pharmacology
037 Drug Literature Index
LA Russian
SL English
CT Medical Descriptors:
*coated vesicle
brain
nerve cell
rat
ultrastructure
animal cell
nonhuman
article
Drug Descriptors:
receptor
***uridine: PD, pharmacology**
RN (uridine) **58-96-8**

L58 ANSWER 13 OF 13 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 88144040 EMBASE
DN 1988144040
TI Effects of short-term administration of cytidine, uridine and levoglutamine, alone or in combination, on the cerebral electrical activity of patients with chronic cerebrovascular disease.
AU Manna V.; Martucci N.
CS Department of Neurology and Neurophysiopathology, Italian Institute of Neurotraumatology, Rome, Italy
SO International Journal of Clinical Pharmacology Research, (1988) 8/3 (199-210).
ISSN: 0251-1649 CODEN: CPHRDE
CY Switzerland
DT Journal
FS 008 Neurology and Neurosurgery
030 Pharmacology
037 Drug Literature Index
LA English
CT Medical Descriptors:
***cerebrovascular disease: DT, drug therapy**

*electroencephalography
human
clinical article
male
female
intravenous drug administration
Drug Descriptors:
***cytidine: DT, drug therapy**
***cytidine: CB, drug combination**
*cytidine: CT, clinical trial
***glutamine: DT, drug therapy**
***glutamine: CB, drug combination**
*glutamine: CT, clinical trial
***uridine: DT, drug therapy**
***uridine: CB, drug combination**
***uridine: CT, clinical trial**
RN (cytidine) 65-46-3; (glutamine) 56-85-9, 6899-04-3; (uridine) **58-96-8**

=> d all tot 159

L59 ANSWER 1 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 1998132764 EMBASE
TI Uridine preserves ATP during hypoxic perfusion of the rat heart.
AU Lin Z.; Richards S.M.; Rosenfeldt F.L.; Pepe S.
CS Z. Lin, Cardiac Surgical Transplant Res Unit, Alfred Hospital, Melbourne, Vic., Australia
SO Asia Pacific Heart Journal, (1997) 6/3 (190-196).
Refs: 30
ISSN: 1328-0163 CODEN: APHJF4
CY Australia
DT Journal; Article
FS 018 Cardiovascular Diseases and Cardiovascular Surgery
037 Drug Literature Index
LA English
SL English
AB Background: The pyrimidine precursor, orotic acid, by minimising ischaemia-induced ATP loss, improves the functional performance of recently infarcted hearts that have been subjected to global ischaemia. However, we have also previously shown that orotic acid is not directly active in the heart but is preferentially taken up by the liver where it is metabolised to uridine. Aim: To investigate whether uridine itself can minimise hypoxia-induced ATP loss. Methods: Isolated Langendorff-mode perfused rat hearts were subjected to 4 protocols after 20 min normoxic stabilisation: normoxia for 30 min (n = 12); hypoxia for 30 min (n = 12); hypoxia in the presence of 17 .mu.M uridine for 30 min (n = 12); and [U-14C]-uridine added directly to the hypoxic perfusate reservoir just prior to 30 min hypoxia (n = 4). [U-14C]-uridine was used to assess the contribution of radiolabel to adenosine formation from adenine nucleotide hydrolysis. Coronary effluent was collected and hearts were freeze-damped for metabolite assay. Results: Hypoxia reduced ATP, from 21.1 .+-. 1.1 to 4.1 .+-. 0.6 .mu.mol/g dry weight ($p < 0.05$), and reduced total adenine nucleotides (TAN) from 30 .+-. 1.2 to 10.2 .+-. 0.9 .mu.mol/g dry weight ($p < 0.05$). Uridine during hypoxia increased myocardial ATP by 94% to 8 .+-. 0.9 .mu.mol/g dry weight and TAN by 50% to 15.3 .+-. 1.1 .mu.mol/g dry weight ($p < 0.05$). Uridine plus hypoxia increased total lactate release by 52% from 768.1 .+-. 8.6 .mu.mol/g dry weight to 1148.4 .+-. 146 .mu.mol/g dry weight compared with hypoxia alone ($p < 0.05$). Although the salvage of purine bases did occur, it was calculated that less than 0.01% of labelled ribose was transferred for salvage of purines. Conclusion: In the present experimental model, uridine protects the hypoxic heart by predominantly enhancing glycolytic energy production.
CT Medical Descriptors:
***heart muscle ischemia**

*heart protection
time
nonhuman
male
rat
controlled study
animal tissue
article
priority journal
Drug Descriptors:
*adenosine triphosphate: EC, endogenous compound
***uridine: PD, pharmacology**
lactic acid: EC, endogenous compound
adenine nucleotide: EC, endogenous compound
orotic acid
RN (adenosine triphosphate) 15237-44-2, 56-65-5, 987-65-5; (uridine) **58-96-8**; (lactic acid) 113-21-3, 50-21-5; (orotic acid) 58915-47-2, 65-86-1

L59 ANSWER 2 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 97324140 EMBASE
DN 1997324140
TI Developmental disorder associated with increased cellular nucleotidase activity.
AU Page T.; Yu A.; Fontanesis J.; Nyhan W.L.
CS T. Page, Department of Neurosciences 0624, Univ. of California at San Diego, San Diego, CA 92093, United States
SO Proceedings of the National Academy of Sciences of the United States of America, (1997) 94/21 (11601-11606).
Refs: 25
ISSN: 0027-8424 CODEN: PNASA6
CY United States
DT Journal; Article
FS 008 Neurology and Neurosurgery
022 Human Genetics
037 Drug Literature Index
LA English
SL English
AB Four unrelated patients are described with a syndrome that included developmental delay, seizures, ataxia, recurrent infections, severe language deficit, and an unusual behavioral phenotype characterized by hyperactivity, short attention span, and poor social interaction. These manifestations appeared within the first few years of life. Each patient displayed abnormalities on EEG. No unusual metabolites were found in plasma or urine, and metabolic testing was normal except for persistent hypouricosuria. Investigation of purine and pyrimidine metabolism in cultured fibroblasts derived from these patients showed normal incorporation of purine bases into nucleotides but decreased incorporation of uridine. De novo synthesis of purines and cellular phosphoribosyl pyrophosphate content also were moderately decreased. The distribution of incorporated purines and pyrimidines did not reveal a pattern suggestive of a deficient enzyme activity. Assay of individual enzymes in fibroblast lysates showed no deficiencies. However, the activity of cytosolic 5'-nucleotidase was elevated 6- to 10-fold. Based on the possibility that the observed increased catabolic activity and decreased pyrimidine salvage might be causing a deficiency of pyrimidine nucleotides, the patients were treated with oral pyrimidine nucleoside or nucleotide compounds. All patients showed remarkable improvement in speech and behavior as well as decreased seizure activity and frequency of infections. A double-blind placebo trial was undertaken to ascertain the efficacy of this supplementation regimen. Upon replacement of the supplements with placebo, all patients showed rapid regression to their pretreatment states. These observations suggest that increased nucleotide catabolism is related to the symptoms of these patients, and that the effects of this increased catabolism are reversed by administration of uridine.
CT Medical Descriptors:

***developmental disorder: DT, drug therapy**
*developmental disorder: CN, congenital disorder
*enzyme activity
article
ataxia: ET, etiology
ataxia: CN, congenital disorder
case report
child
clinical feature
clinical trial
controlled study
diet supplementation
double blind procedure
electroencephalogram
female
human
human cell
hyperactivity: ET, etiology
language disability: ET, etiology
male
nucleotide metabolism
priority journal
purine metabolism
pyrimidine metabolism
randomized controlled trial
recurrent infection: ET, etiology
seizure: ET, etiology
seizure: CN, congenital disorder
Drug Descriptors:
*nucleotidase
*purine
*pyrimidine
***uridine: DO, drug dose**
***uridine: DT, drug therapy**
phosphoribosyl pyrophosphate
pyrimidine nucleoside
RN (nucleotidase) 9033-33-4; (purine) 120-73-0; (pyrimidine) 289-95-2; (uridine) **58-96-8**; (phosphoribosyl pyrophosphate) 7540-64-9

L59 ANSWER 3 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 97255450 EMBASE
DN 1997255450
TI A longitudinal study of cognitive functioning in patients with classical galactosaemia, including a cohort treated with oral uridine.
AU Manis F.R.; Cohn L.B.; McBride-Chang C.; Wolff J.A.; Kaufman F.R.
CS F.R. Manis, Department of Psychology, University of Southern California, Los Angeles, CA 90089-1061, United States
SO Journal of Inherited Metabolic Disease, (1997) 20/4 (549-555).
Refs: 15
ISSN: 0141-8955 CODEN: JIMDDP
CY Netherlands
DT Journal; Article
FS 007 Pediatrics and Pediatric Surgery
008 Neurology and Neurosurgery
029 Clinical Biochemistry
037 Drug Literature Index
LA English
SL English
AB Existing longitudinal data on patients with classical galactosaemia suggests that neurocognitive functioning is impaired and, in isolated case reports, may show a decline in performance over time. The present study explored whether there are long-term changes in cognitive abilities in patients with galactosaemia and whether oral uridine can improve neurocognitive performance. Thirty-five patients (18 males, 17 females), 29 of whom received oral uridine powder at 150 mg/kg per day (divided dose, three times daily), were evaluated over a 2-5-year period with the

Woodcock-Johnson Revised Cognitive Abilities Test, three academic achievement tests, and the Beery Test of Visual Motor Integration. Results showed that the uridine cohort and a comparison group that received only dietary restriction made small gains in cognitive performance over the treatment period and the size of the gains did nor differ significantly. Seven subjects who started uridine prior to the age of 14 months did not differ significantly in their cognitive test scores at an average age of 3.5 years from a group of older children who had begun treatment at 4.5 years of age. These results provide no support for any developmental or uridine-treatment-related change in cognitive functioning for this sample of galactosaemic subjects.

CT Medical Descriptors:
***cognition**
***galactosemia: DT, drug therapy**
*galactosemia: TH, therapy
adolescent
article
child
clinical article
controlled study
diet restriction
female
human
infant
longitudinal study
male
motor activity
newborn
oral drug administration
powder
task performance
Drug Descriptors:
*galactose: EC, endogenous compound
***uridine: AD, drug administration**
***uridine: DO, drug dose**
***uridine: DT, drug therapy**
***uridine: PR, pharmaceutics**

RN (galactose) 26566-61-0, 50855-33-9, 59-23-4; (uridine) **58-96-8**

L59 ANSWER 4 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 97023727 EMBASE
DN 1997023727
TI Guidelines for symptomatic therapy in spinal muscular atrophy (SMA).
AU Zerres K.; Rudnik-Schoneborn S.; Dubowitz V.; Emery A.E.H.; Forst R.; Granata C.; Haverkamp F.; Merlini L.; Mielke U.; Mokrusch T.; Mortier W.; Nix W.A.; Rudel R.; Voit T.; Wollinsky K.H.; Zierz S.; Rohrig D.
CS Prof. Dr. K. Zerres, Institut fur Humangenetik, Universitat Bonn, Wilhelmstr. 31, D-53111 Bonn, Germany
SO Acta Cardiomiologica, (1995) 7/2 (61-66).
Refs: 25
ISSN: 1124-8874 CODEN: ACCAFV
CY Italy
DT Journal; (Short Survey)
FS 008 Neurology and Neurosurgery
019 Rehabilitation and Physical Medicine
022 Human Genetics
037 Drug Literature Index
LA English
SL English
AB Recommendations for symptomatic treatment of proximal spinal muscular atrophy (SMA) are still controversial. As there is as yet no therapeutic strategy leading to functional recovery in SMA, symptomatic therapies play a major role in the medical care of SMA patients. Therefore a workshop was initiated to propose common guidelines for physiotherapy, orthopedic treatment and ventilatory support in SMA. It was concluded that in severe SMA I, overactivity has to be avoided and that ventilatory support should

be offered under certain limited circumstances only, although the overall prognosis is poor. In intermediate SMA II and mild SMA III, active physiotherapy is useful to prevent contractures and to maintain mobility as long as possibile. Orthopedic treatment in different disease stages is important and includes surgical procedures of limbs and spine. Patients with SMA II and III should receive all available ventilatory support, starting with preventive respiratory training and proceeding to assisted ventilation, if hypoventilation becomes evident. According to prevailing belief, neither electrical stimulation nor drug treatment can currently be recommended in SMA.

CT Medical Descriptors:
***spinal muscular atrophy: TH, therapy**
***spinal muscular atrophy: RH, rehabilitation**
artificial ventilation
human
orthopedics
physiotherapy
short survey
Drug Descriptors:
***azathioprine: DT, drug therapy**
***corticosteroid: DT, drug therapy**
***corticotropin: DT, drug therapy**
***ganglioside: DT, drug therapy**
***orotic acid: DT, drug therapy**
***physostigmine: DT, drug therapy**
***tacrine: DT, drug therapy**
***uridine: DT, drug therapy**

RN (azathioprine) 446-86-6; (corticotropin) 11136-52-0, 9002-60-2, 9061-27-2; (orotic acid) 58915-47-2, 65-86-1; (physostigmine) 57-47-6, 64-47-1; (tacrine) 1684-40-8, 3198-41-2, 321-64-2; (uridine) **58-96-8**

L59 ANSWER 5 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 96329473 EMBASE
DN 1996329473
TI A Phase I trial of a modified, dose intensive FAMTX regimen (high dose 5-fluorouracil + doxorubicin + high dose methotrexate + leucovorin) with oral uridine rescue.
AU Schwartz G.K.; Christman K.; Saltz L.; Casper E.; Quan V.; Bertino J.; Martin D.S.; Colofiore J.; Kelsen D.
CS Memorial Sloan-Kettering Cancer Ctr., 1275 New York Ave.,New York, NY 10021, United States
SO Cancer, (1996) 78/9 (1988-1995).
ISSN: 0008-543X CODEN: CANCAR
CY United States
DT Journal; Article
FS 016 Cancer
037 Drug Literature Index
048 Gastroenterology
LA English
SL English
AB BACKGROUND. Dose intensification of 5-fluorouracil (5-FU) is complicated by increased toxicity, 5-FU is a fluorine-substitute analog of uracil. In preclinical studies, administration of oral uridine (Ur) has been shown to allow for dose intensification of 5-Fu with enhancement of its antitumor activity. Therefore, a Phase I trial was designed aimed at dose intensification of 5-FU as a component of a modified 5-FU-doxorubicin-methotrexate (FAMTX) regimen using oral Ur rescue. METHODS. Methotrexate (MTX) was administered to all patients at a fixed dose of 1.5 g/m2. MTX was followed 24 hours later by escalating doses of 5-FU starting at 800 mg/m2 with leucovorin rescue. Cycles of 5-FU and MTX were repeated every 15 days. Every other cycle, patients received doxorubicin ('Adria cycles') at a dose of 30 mg/m2. Oral Ur was administered at a dose of 8 mg/m2 every 6 hours for 12 doses. In the first phase of the study, patients received Ur only if they developed Grade 3 or 4 hematologic toxicity. In the second phase, all patients received Ur 24 hours after 5-FU on all cycles. RESULTS. Without Ur rescue, the maximum tolerated dose (MTD) of 5-FU was

900 mg/m2 on the Adria cycles and 1.1 mg/m2 on the non-adria cycles. With Ur, the MTD of 5-FU increased to 1.2 mg/m2 on the adria cycles and to 1.6 mg/m2 on the non- adria cycles. CONCLUSIONS. In this modified FAMTX regimen, oral Ur administration allowed for dose-intensification of 5-FU, with a 33% increase in the MTD of 5-FU on the Adria cycles and a 45% increase in the MTD of 5-FU dose on the non-Adria cycles.

CT Medical Descriptors:
*stomach carcinoma: DI, diagnosis
***stomach carcinoma: DT, drug therapy**
adult
antineoplastic activity
article
cancer control
cancer survival
clinical trial
controlled study
dose response
drug efficacy
drug tolerance
human
major clinical study
oral drug administration
patient compliance
phase 1 clinical trial
priority journal
Drug Descriptors:
*doxorubicin: CT, clinical trial
***doxorubicin: CB, drug combination**
*doxorubicin: DO, drug dose
***doxorubicin: DT, drug therapy**
***fluorouracil: CB, drug combination**
*fluorouracil: DO, drug dose
***fluorouracil: DT, drug therapy**
*fluorouracil: CT, clinical trial
*folinic acid: CT, clinical trial
***folinic acid: DT, drug therapy**
*folinic acid: DO, drug dose
***folinic acid: CB, drug combination**
***methotrexate: CB, drug combination**
***methotrexate: DT, drug therapy**
*methotrexate: DO, drug dose
*methotrexate: CT, clinical trial
***uridine derivative: CT, clinical trial**
***uridine derivative: DO, drug dose**
***uridine derivative: DT, drug therapy**
***uridine derivative: PK, pharmacokinetics**

RN (doxorubicin) 23214-92-8, 25316-40-9; (fluorouracil) 51-21-8; (folinic acid) 58-05-9, 68538-85-2; (methotrexate) 15475-56-6, 59-05-2, 7413-34-5

L59 ANSWER 6 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 96303055 EMBASE
DN 1996303055
TI N3-Phenacyluridine, a novel hypnotic compound, interacts with the benzodiazepine receptor.
AU Kimura T.; Kuze J.; Watanabe K.; Kondo S.; Ho I.K.; Yamamoto I.
CS Department of Hygienic Chemistry, Faculty of Pharmaceutical Sciences, Hokuriku University, 3-Ho, Kanagawa-machi,Kanazawa 920-11, Japan
SO European Journal of Pharmacology, (1996) 311/2-3 (265-269).
ISSN: 0014-2999 CODEN: EJPHAZ
CY Netherlands
DT Journal; Article
FS 008 Neurology and Neurosurgery
030 Pharmacology
032 Psychiatry
037 Drug Literature Index
LA English

SL English
AB N3-Phenacyluridine (3-phenacyl-1-.beta.-D-ribofuranosyluracil) has potent sedative and hypnotic activities following intracerebroventricular injection in mice. To study the mechanism of action of N3-phenacyluridine, the interaction of this compound with the benzodiazepine receptor has been investigated. Results obtained showed that this compound inhibited specific binding of [3H]flunitrazepam to synaptic membranes of bovine cortex in a concentration-dependent fashion (IC50 = 129 .mu.M). Scatchard analysis of [3H]flunitrazepam binding revealed that N3-phenacyluridine interacted with the ligand at the benzodiazepine receptor binding site in a competitive manner. Ro 15-1788 (8-fluoro-3-carboethoxy-5,6-dihydro-5-methyl-6-oxo-4H- imidazo[1,5a]1,4-benzodiazepine), a benzodiazepine receptor antagonist, also inhibited the specific binding of [3H]flunitrazepam in the presence of the compound. The results suggest that the pharmacological activity of N3-phenacyluridine may be partially mediated through the benzodiazepine receptor.
CT Medical Descriptors:
***brain cortex**
***synaptic membrane**
animal tissue
article
binding site
cattle
controlled study
drug receptor binding
nonhuman
priority journal
Drug Descriptors:
***benzodiazepine receptor: EC, endogenous compound**
***hypnotic agent: CM, drug comparison**
*hypnotic agent: AN, drug analysis
***hypnotic agent: PD, pharmacology**
*hypnotic agent: DO, drug dose
*hypnotic agent: DV, drug development
***uridine derivative: DO, drug dose**
***uridine derivative: PD, pharmacology**
***uridine derivative: CM, drug comparison**
***uridine derivative: AN, drug analysis**
***uridine derivative: DV, drug development**
benzodiazepine receptor blocking agent: PD, pharmacology
benzodiazepine receptor blocking agent: DO, drug dose
benzodiazepine receptor blocking agent: CM, drug comparison
flumazenil: PD, pharmacology
flumazenil: DO, drug dose
flumazenil: CM, drug comparison
flunitrazepam: CM, drug comparison
RN (flumazenil) 78755-81-4; (flunitrazepam) 1622-62-4
CN (1) Ro 151788
CO (1) Hoffmann la roche (Japan)

L59 ANSWER 7 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 95342810 EMBASE
DN 1995342810
TI Uridine reduces rotation induced by L-dopa and methamphetamine in 6-OHDA-treated rats.
AU Myers C.S.; Fisher H.; Wagner G.C.
CS Department of Psychology, Busch Campus, Rutgers University,New Brunswick, NJ 08903, United States
SO Pharmacology Biochemistry and Behavior, (1995) 52/4 (749-753).
ISSN: 0091-3057 CODEN: PBBHAU
CY United States
DT Journal; Article
FS 008 Neurology and Neurosurgery
030 Pharmacology
037 Drug Literature Index
LA English

SL English

AB The pyrimidine nucleoside uridine may reduce side effects associated with antipsychotic medication by interacting with dopamine or GABA neurotransmission. Male Sprague-Dawley rats were used to investigate coadministration of uridine with agents that alter food intake (amphetamine, haloperidol, and chlordiazepoxide) and locomotor activity (methamphetamine and L-dopa). Results indicated that chronic uridine [32.0 mg/kg, intraperitoneally (IP)] alone did not alter milk intake or reduction of milk intake induced by amphetamine (dose range 0.5-2.0 mg/kg, IP) or haloperidol (0.125-1.0 mg/kg, IP), nor did it alter the biphasic response induced by chlordiazepoxide (5.0-40.0 mg/kg, IP). However, uridine-treated animals with unilateral striatal lesions exhibited no rotational behavior in the absence of drug challenge, but showed decreased rotation induced by the dopamine agonist, L-dopa (50.0-200.0 mg/kg, IP) compared with controls. In addition, uridine-treated rats exhibited reduced rotation after repeated injections of methamphetamine (4.0 mg/kg, IP) in contrast to increasingly greater rotation observed in control animals. These results are further evidence that chronic uridine may alter drug-induced dopaminergic activity without exerting effects itself.

CT Medical Descriptors:
***brain injury**
***circling behavior**
***corpus striatum**
***dopaminergic transmission**
animal experiment
animal model
article
controlled study
drug antagonism
food intake
gabaergic transmission
intraperitoneal drug administration
locomotion
male
nonhuman
priority journal
rat
Drug Descriptors:
milk
*levodopa: DO, drug dose
***levodopa: CB, drug combination**
***levodopa: IT, drug interaction**
***levodopa: PD, pharmacology**
*methamphetamine: DO, drug dose
***methamphetamine: IT, drug interaction**
***methamphetamine: PD, pharmacology**
***uridine: PD, pharmacology**
***uridine: IT, drug interaction**
amphetamine: DO, drug dose
amphetamine: PD, pharmacology
benserazide: CB, drug combination
benserazide: PD, pharmacology
chlordiazepoxide: DO, drug dose
chlordiazepoxide: PD, pharmacology
dopamine receptor stimulating agent: IT, drug interaction
dopamine receptor stimulating agent: DO, drug dose
dopamine receptor stimulating agent: PD, pharmacology
haloperidol: PD, pharmacology
haloperidol: DO, drug dose
oxidopamine: TO, drug toxicity
pyrimidine nucleoside: IT, drug interaction
pyrimidine nucleoside: PD, pharmacology

RN (milk) 8049-98-7; (levodopa) 59-92-7; (methamphetamine) 28297-73-6, 51-57-0, 537-46-2, 7632-10-2; (uridine) **58-96-8**; (amphetamine) 1200-47-1, 139-10-6, 156-34-3, 2706-50-5, 300-62-9, 51-62-7, 60-13-9, 60-15-1; (benserazide) 14919-77-8, 322-35-0; (chlordiazepoxide) 438-41-5,

58-25-3; (haloperidol) 52-86-8; (oxidopamine) 1199-18-4, 28094-15-7, 636-00-0
CN Ro 4 4602

L59 ANSWER 8 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 94380783 EMBASE
DN 1994380783
TI Sleep promoting effects of N3-benzyluridine in unrestrained rats.
AU Kimura T.; Watanabe K.; Yamamoto I.; Honda K.; Inoue S.
CS Department of Hygienic Chemistry, Faculty of Pharmaceutical Sciences, Hokuriku University, 3-Ho, Kanagawa-machi,Kanazawa 920-11, Japan
SO Research Communications in Psychology, Psychiatry and Behavior, (1993) 18/3-4 (111-119).
ISSN: 0362-2428 CODEN: RCPBDC
CY United States
DT Journal; Article
FS 002 Physiology
030 Pharmacology
032 Psychiatry
037 Drug Literature Index
LA English
SL English
AB Effect of N3-benzyluridine (N3-ByUd), having hypnotic activity, on rat natural sleep was studied. When N3-ByUd (1 and 10 pmol) was nocturnally infused to rat third ventricle from 19:00 to 05:00 in unrestrained environment, rat sleep was significantly increased compared to saline infused rats during light period of recovery day. Total time and frequency of paradoxical sleep (PS) of rats infused with N3-ByUd (1 pmol/10 h) during light period of recovery day increased 22 and 25%, respectively, compared to that of control (saline). This compound (10 pmol/10 h) also increased in frequency of slow wave sleep (SWS) by 119% of control. These results indicate that N3-ByUd affects at least in part the natural sleep of rats.
CT Medical Descriptors:
*circadian rhythm
***sleep pattern**
animal experiment
article
controlled study
drug synthesis
electroencephalography
electromyography
intracerebroventricular drug administration
male
nonhuman
rat
rem sleep
slow wave sleep
Drug Descriptors:
***uridine derivative: DV, drug development**
***uridine derivative: PD, pharmacology**
3 benzyluridine: DV, drug development
3 benzyluridine: PD, pharmacology
pentobarbital
reagent
uridine
unclassified drug
RN (pentobarbital) 57-33-0, 76-74-4; (uridine) **58-96-8**
CO Abbott (United States); Kohjin (Japan); Wako pure chemical industry (Japan)

L59 ANSWER 9 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 94219765 EMBASE
DN 1994219765
TI Uridine potentiates haloperidol's disruption of conditioned avoidance responding.

AU Myers C.S.; Fisher H.; Wagner G.C.
CS Department of Psychology, Busch Campus, Rutgers University,New Brunswick, NJ 08903, United States
SO Brain Research, (1994) 651/1-2 (194-198).
ISSN: 0006-8993 CODEN: BRREAP
CY Netherlands
DT Journal; Article
FS 002 Physiology
008 Neurology and Neurosurgery
032 Psychiatry
030 Pharmacology
037 Drug Literature Index
LA English
SL English
AB The pyrimidine nucleoside, uridine, has been proposed as a potential supplement in the treatment of psychosis based on its ability to reduce haloperidol-induced dopamine release. These experiments investigated the effect of uridine (32 mg/kg, i.p.) coadministered with the neuroleptic haloperidol, on rats engaged in one way conditioned avoidance responding. Uridine itself had no effect on animals' performance, while haloperidol (dose range 0.05-0.4 mg/kg, i.p., 90 min before test session) decreased number of avoidances and increased avoidance and escape latencies in a dose-dependent manner. When coadministered with haloperidol, uridine significantly potentiated the disruption of avoidance and avoidance latency induced by haloperidol. This potentiation was still evident after chronic (27 days) uridine treatment. Importantly, coadministration of uridine did not potentiate haloperidol-induced increase of escape latency. The potentiation of haloperidol-induced disruption of conditioned avoidance responding suggests that uridine coadministration might enhance the antipsychotic action of traditional neuroleptics. This would allow for a reduction in the therapeutic dose of the antipsychotic, thereby reducing side effect frequency.
CT Medical Descriptors:
***psychosis: DT, drug therapy**
animal experiment
article
avoidance behavior
conditioning
controlled study
intraperitoneal drug administration
male
nonhuman
priority journal
rat
Drug Descriptors:
***haloperidol: PD, pharmacology**
***haloperidol: DT, drug therapy**
*haloperidol: DO, drug dose
***haloperidol: IT, drug interaction**
***haloperidol: CB, drug combination**
***uridine: PD, pharmacology**
***uridine: IT, drug interaction**
***uridine: CB, drug combination**
nucleoside
RN (haloperidol) 52-86-8; (uridine) **58-96-8**

L59 ANSWER 10 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 94172847 EMBASE
DN 1994172847
TI Boron neutron capture therapy of primary and metastatic brain tumors.
AU Barth R.F.; Soloway A.H.
CS Department of Pathology, Ohio State University,Columbus, OH 43210, United States
SO Molecular and Chemical Neuropathology, (1994) 21/2-3 (139-154).
ISSN: 1044-7393 CODEN: MCHNEM
CY United States

DT Journal; Conference Article
FS 008 Neurology and Neurosurgery
016 Cancer
023 Nuclear Medicine
037 Drug Literature Index
LA English
SL English
AB Boron neutron capture therapy (BNCT) is based on the nuclear reaction that occurs when a stable isotope, boron-10, is irradiated with low energy (0.025 eV) thermal neutrons (n(th)) to yield alpha (4He) particles and 7Li nuclei (10B + n(th) .fwdarw. [11B] .fwdarw. 4He + 7Li + 2.79 MeV). The success of BNCT as a tumoricidal modality is dependent on the delivery of a sufficient quantity of 10B and n(th) to individual cancer cells to sustain a lethal 10B(n, .alpha.) 7Li reaction. Boron delivery agents include a variety of compounds, such as the sulfhydryl containing polyhedral borane sodium borocaptate (Na2B12H11SH, [BSH]), boronoporphyrins, boronophenylalanine, carboranyl uridines (CBU), and boronated monoclonal antibodies (MAb). The present review will focus on three delivery systems that currently are under investigation in our laboratories, boronated monoclonal antibodies, carboranyl uridines, and boronophenylalanine. Methodology has been developed to heavily boronate MAb using a precision macromolecule, a 'starburst' dendrimer, which can be linked to MAb by means of heterobifunctional reagents. Although the resulting immunoconjugates retain their in vitro immunoreactivity, they lose their in vivo tumor localizing properties and accumulate in the liver. In order to obviate this problem, work is now in progress to produce bispecific MAb, which can simultaneously recognize a tumor-associated antigen and a boronated macromolecule. Boron containing nucleosides are potential vehicles for incorporating boron compounds into nucleic acids of neoplastic cells. For this purpose, carboranyl uridines have been synthesized with the boron moiety on either the pyrimidine base or on the carbohydrate component. Although such structures appear to be avidly taken up and retained by tumor cells in vitro, only the 5-carboranyl-nucleosides are converted biologically to the nucleotide. There is no evidence, however, that the latter are incorporated into nucleic acids. Other carboranyl nucleosides currently are being synthesized that may have better tumor localizing properties. The potential use of boronophenylalanine as a capture agent for the treatment of melanoma metastatic to the brain also is under investigation. A nude rat model has been developed using human melanoma cells that are stereotactically implanted into the brain. BNCT-treated animals have either had prolonged survival times or continue to live compared to control rats that invariably died of their tumors, thereby suggesting therapeutic efficacy.
CT Medical Descriptors:
***brain metastasis: RT, radiotherapy**
***brain tumor: RT, radiotherapy**
cancer survival
clinical trial
conference paper
drug conjugation
human
intralesional drug administration
intraperitoneal drug administration
intravenous drug administration
melanoma: RT, radiotherapy
neutron capture therapy
nonhuman
priority journal
tumor localization
Drug Descriptors:
*boronic acid derivative: DV, drug development
*boronic acid derivative: PR, pharmaceutics
*monoclonal antibody: PR, pharmaceutics
*monoclonal antibody: DV, drug development
*nucleoside derivative: DV, drug development

*nucleoside derivative: PR, pharmaceutics
***uridine derivative: PR, pharmaceutics**
***uridine derivative: DV, drug development**
4 boronophenylalanine: PR, pharmaceutics
borane derivative: PR, pharmaceutics
borocaptate sodium: PR, pharmaceutics
boron 10
boronoporphyrin derivative: PR, pharmaceutics
carboranylalanine: PR, pharmaceutics
deuteroporphyrin derivative: PR, pharmaceutics
unclassified drug
RN (4 boronophenylalanine) 76410-58-7; (boron 10) 14798-12-0; (carboranylalanine) 61216-60-2

L59 ANSWER 11 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 94166153 EMBASE
DN 1994166153
TI [Effects of a nucleotide-nucleoside mixture on the ischemic muscular metabolism in patients with peripheral arterial occlusive disease stage II].
EFFEKTE EINES NUKLEOTID-NUKLEOSID-GEMISCHES AUF DEN ISCHAMISCHEN MUSKELSTOFFWECHSEL BEI PATIENTEN MIT PERIPHERER ARTERIELLER VERSCHLUSSKRANKHEIT STADIUM II..
AU Rexroth W.; Huber K.H.; Radle J.; Semmler W.; Van Kaick G.
CS Abteilung Innere Medizin, St. Josefskrankenhaus, Landhausstrasse 25,D-69115 Heidelberg, Germany
SO Vasa - Journal of Vascular Diseases, (1994) 23/2 (98-108).
ISSN: 0301-1526 CODEN: VASAAH
CY Switzerland
DT Journal; Article
FS 008 Neurology and Neurosurgery
023 Nuclear Medicine
037 Drug Literature Index
LA German
SL German; English
AB In order to assess the acute metabolic effects of an intraarterial infusion of nucleotide-nucleoside-mixture (NNM), 31P-mr-spectroscopy at the site of m. gastrocnemius and metabolite determinations from blood of the femoral artery and vein were carried out in 10 patients with PAOD stage II during ergometric calf exercise to the claudication pain limit. The spectroscopic measurements revealed a greater exercise-induced fall of PCr and a higher increase of P(i) in calf muscles during supply of NNM compared with control ergometry. Post-exercise recovery of PCr was distinctly delayed during infusion of NNM. The anaerobic production of energy, however, was sufficient to maintain the ATP concentration to the same extent as under control ergometry. On the other hand, intramuscular lactate acidosis developed to a lower degree with NNM infusion than without NNM. A reduced muscular release of lactate, pyruvate, ammonia and alanine followed from the evaluation of the arteriovenous balance of these metabolites in the femoral vessels indicating a favourable global metabolic effect of NNM infusion in the extremity. The apparent contradiction in the spectroscopic and analytic-biochemical findings can be explained by local blood shunts induced by maximum vasodilation. Noninvasive mr-spectroscopy allows to detect directly and continuously the metabolic impact of ischemia in the calf muscles afflicted by arterial occlusion, whereas the metabolite concentrations in femoral blood are altered by afflux from non-ischemic areas. The known clinical benefit of frequently repeated intraarterial infusions of NNM is thought to be due to an expansion of collateral circulation and to a favourable influence on endothelial functions.
CT Medical Descriptors:
*collateral circulation
***intermittent claudication: DT, drug therapy**
***muscle ischemia**
*muscle metabolism
*peripheral occlusive artery disease

adult
aged
angiography
article
clinical article
clinical trial
drug mixture
exercise
human
intraarterial drug administration
male
phosphorus nuclear magnetic resonance
Drug Descriptors:
***adenosine diphosphate: CB, drug combination**
***adenosine diphosphate: DT, drug therapy**
***adenosine diphosphate: PD, pharmacology**
***adenosine phosphate: DT, drug therapy**
***adenosine phosphate: CB, drug combination**
***adenosine phosphate: PD, pharmacology**
***adenosine triphosphate: PD, pharmacology**
***adenosine triphosphate: DT, drug therapy**
***adenosine triphosphate: CB, drug combination**
***guanosine: PD, pharmacology**
***guanosine: DT, drug therapy**
***guanosine: CB, drug combination**
***guanosine phosphate: DT, drug therapy**
***guanosine phosphate: CB, drug combination**
***guanosine phosphate: PD, pharmacology**
***inosine: PD, pharmacology**
***inosine: DT, drug therapy**
***inosine: CB, drug combination**
***nucleoside: CB, drug combination**
***nucleoside: DT, drug therapy**
***nucleoside: PD, pharmacology**
***nucleotide: PD, pharmacology**
***nucleotide: CB, drug combination**
***nucleotide: DT, drug therapy**
***uridine: PD, pharmacology**
***uridine: DT, drug therapy**
***uridine: CB, drug combination**
laevadosin: PD, pharmacology
laevadosin: DT, drug therapy
unclassified drug
RN (adenosine diphosphate) 20398-34-9, 58-64-0; (adenosine phosphate) 61-19-8, 8063-98-7; (adenosine triphosphate) 15237-44-2, 56-65-5, 987-65-5; (guanosine) 118-00-3; (guanosine phosphate) 29593-02-0, 5550-12-9, 85-32-5; (inosine) 58-63-9; (uridine) **58-96-8**; (laevadosin) 60675-64-1
CN Laevadosin

L59 ANSWER 12 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 94144034 EMBASE
DN 1994144034
TI The potent depressant effects of N3-phenacyluridine in mice.
AU Yamamoto I.; Kuze J.; Kimura T.; Watanabe K.; Kondo S.; Ho I.K.
CS Faculty of Pharmaceutical Sciences, Hokuriku University, 3-Ho, Kanagawa-machi,Kanazawa 920-11, Japan
SO Biological and Pharmaceutical Bulletin, (1994) 17/4 (514-516). ISSN: 0918-6158 CODEN: BPBLEO
CY Japan
DT Journal; Article
FS 008 Neurology and Neurosurgery
032 Psychiatry
030 Pharmacology
037 Drug Literature Index
LA English

SL English
AB The hypnotic activity of N3-phenacyluridine in 2.0 .mu.mol/mouse by intracerebroventricular (i.c.v.) injection was 20 times stronger than that of known N3-benzyluridine. In 0.5 .mu.mol/mouse, i.c.v., this compound strongly potentiated both pentobarbital- and diazepam-induced sleep as compared to N3-substituted uridines, including N3-benzyluridine. Furthermore, the compound caused motor incoordination as well as decreasing spontaneous activity in the same dose. These results indicate that among the N3-substituted uridines and related compounds previously reported, N3-phenacyluridine possesses potent depressant effects.
CT Medical Descriptors:
***central nervous system depression**
***hypnosis**
***sleep time**
animal experiment
article
behavior
controlled study
intracerebroventricular drug administration
locomotion
male
mouse
nonhuman
Drug Descriptors:
***uridine derivative: CM, drug comparison**
***uridine derivative: CB, drug combination**
***uridine derivative: IT, drug interaction**
***uridine derivative: PD, pharmacology**
3 n benzyluridine: PD, pharmacology
3 n benzyluridine: CB, drug combination
3 n benzyluridine: IT, drug interaction
3 n benzyluridine: CM, drug comparison
3 n phenacyluridine: CM, drug comparison
3 n phenacyluridine: IT, drug interaction
3 n phenacyluridine: PD, pharmacology
3 n phenacyluridine: DV, drug development
3 n phenacyluridine: AN, drug analysis
3 n phenacyluridine: CB, drug combination
diazepam: CB, drug combination
diazepam: IT, drug interaction
diazepam: PD, pharmacology
pentobarbital: CB, drug combination
pentobarbital: IT, drug interaction
pentobarbital: PD, pharmacology
unclassified drug
RN (diazepam) 439-14-5; (pentobarbital) 57-33-0, 76-74-4
CO Kasei (Japan); Yamanouchi pharmaceutical (Japan); Yamasa shoyu (Japan)

L59 ANSWER 13 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 93213059 EMBASE
DN 1993213059
TI Lateral preoptic lesions void slow-wave sleep enhanced by uridine but not by muramyl dipeptide in rats.
AU Kimura-Takeuchi M.; Inoue S.
CS Inst. for Medical/Dental Engineering, Tokyo Medical and Dental University, 2-3-10 Surugadai Kandai,Chiyoda-ku, Tokyo 101, Japan
SO Neuroscience Letters, (1993) 157/1 (17-20).
ISSN: 0304-3940 CODEN: NELED5
CY Ireland
DT Journal; Article
FS 002 Physiology
008 Neurology and Neurosurgery
030 Pharmacology
037 Drug Literature Index
LA English
SL English

AB We investigated the site of action of two sleep-inducing substances, viz., muramyl dipeptide (MDP) and uridine. Localized electrolytic lesions were made bilaterally in the lateral preoptic hypothalamus (LPO) in rats and nocturnal 10-h i.c.v. infusions of MDP and uridine were performed before and after the LPO lesions. MDP increased only slow-wave sleep (SWS) in both intact and LPO-lesioned rats. Uridine promoted both SWS and paradoxical sleep (PS) before the LPO lesions whereas it increased only PS after the lesions. These results suggest that the LPO is crucial for SWS-promoting action of uridine but not MDP.

CT Medical Descriptors:
***brain injury**
***preoptic area**
***slow wave sleep**
animal experiment
animal tissue
article
brain temperature
controlled study
intravenous drug administration
nonhuman
priority journal
rat
rem sleep
Drug Descriptors:
***muramyl dipeptide: PD, pharmacology**
***uridine: PD, pharmacology**

RN (uridine) **58-96-8**

L59 ANSWER 14 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 93210055 EMBASE
DN 1993210055
TI Interaction of the mioflazine derivtive R75231 with the nucleoside transporter: Evidence for positive cooperativity.
AU Jones K.W.; Hammond J.R.
CS Department Pharmacology/Toxicology, Medical Science Building, University of Western Ontario,London, Ont. N6A 5C1, Canada
SO European Journal of Pharmacology - Molecular Pharmacology Section, (1993) 246/2 (97-104).
ISSN: 0922-4106 CODEN: EJPPET
CY Netherlands
DT Journal; Article
FS 023 Nuclear Medicine
029 Clinical Biochemistry
030 Pharmacology
037 Drug Literature Index
LA English
SL English
AB This study investigated the interaction of the mioflazine derivative R75231 with the nucleoside transport system of rabbit cortical synaptosomes, and assessed the binding of [3H]R75231 to human erythrocyte ghost membranes. R75231 was a potent inhibitor of [3H]nitrobenzylthioinosine binding and [3H]uridine uptake in synaptosomes (K(i) < 10 nM). This inhibition was evident even after extensive washing of the synaptosomes, subsequent to exposure to R75231. In addition to its tight binding characteristics, R75231 was shown to be a 'mixed'type inhibitor of [3H]nitrobenzylthioinosine binding (increased K(D), decresed B(max)). [3H]R75231 bound with high affinity (K(D) = 0.4 nM) to erythrocyte membranes wiht a B(max) of 44 pmol/mg protein, which is comparable to the number of [3H]nitrobenyzylthioinosien binding sites in this preparation. Binding of [3H]R75231 to these membranes was reversible, but the rate of dissociation was dependent upon the displacer used. Nitrobenzylthioinosine and dipyridamole each induced a complete dissociation of site-bound [3H]R75231 at rates not significantly different from those observed using a protocol involving a 100-fold dilution with buffer (no displacer). However, U75231 and mioflazine slowed the rate of dissociation of [3H]R75231 and actually caused an initial increase in the

amount of site-bound [3H]R75231. Adenosine, on the other hand, enhanced the rate of [3H]R75231 dissociation. These results indicate that R75231 binding to the nucleoside transporter is a complex reaction, which may involve multiple interacting sites demonstrating positive cooperativity.

CT Medical Descriptors:
***brain cortex**
*erythrocyte membrane
*nucleoside transport
animal tissue
article
binding affinity
brain synaptosome
concentration response
controlled study
dissociation constant
human
human tissue
nonhuman
priority journal
rabbit
Drug Descriptors:
***adenosine: PD, pharmacology**
***dipyridamole: PD, pharmacology**
***draflazine: PD, pharmacology**
***mioflazine: PD, pharmacology**
***nitrobenzylthioinosine: PD, pharmacology**
***uridine: PK, pharmacokinetics**
dilazep: PD, pharmacology
nitrobenzylthioguanosine: PD, pharmacology
radioisotope

RN (adenosine) 58-61-7; (dipyridamole) 58-32-2; (draflazine) 120770-34-5; (mioflazine) 79467-23-5, 83898-67-3; (nitrobenzylthioinosine) 65177-80-2; (uridine) **58-96-8**; (dilazep) 20153-98-4, 35898-87-4; (nitrobenzylthioguanosine) 13153-27-0

CN (1) R 75231

CO (1) Janssen (Belgium); Icn (Canada); Asta (Germany); Sigma (United States); Moravek (United States)

L59 ANSWER 15 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.

AN 93102822 EMBASE

DN 1993102822

TI Differential sleep modulation by sequentially administered muramyl dipeptide and uridine.

AU Kimura-Takeuchi M.; Inoue S.

CS Medical/Dental Engineering Institute, Tokyo Medical and Dental University, Kanda-Surugadai 2-3-10,Chiyoda-ku, Tokyo 101, Japan

SO Brain Research Bulletin, (1993) 31/1-2 (33-37).
ISSN: 0361-9230 CODEN: BRBUDU

CY United States

DT Journal; Article

FS 002 Physiology
008 Neurology and Neurosurgery
030 Pharmacology
037 Drug Literature Index

LA English

SL English

AB In an attempt to investigate the effects on sleep modulation of order of administration of two substances, sequential intracerebroventricular (ICV) infusions of muramyl dipeptide (MDP, 1.0 nmol) and uridine (5.0 pmol) were conducted in freely behaving rats. To eliminate the influence of intermittent infusions on their behavior, the rats were continuously ICV infused with physiological saline solution, which did not affect their normal sleep- waking dynamics. Under these experimental conditions, uridine infusion (2400- 0500 h) attenuated the enhancement of slow-wave sleep (SWS) caused by prior infusion of MDP (1900-2400 h) to the baseline level and, thus, did not exert a sleep-promoting property. In contrast,

MDP infusion (2400-0500 h) further potentiated the SWS-enhancing activity of preinfused uridine (1900-2400 h). A single infusion of MDP or uridine (2400-0500 h) similarly enhanced SWS. These results demonstrate that observed differences in the induction and maintenance of sleep are dependent upon the order of exogenously infused uridine and MDP. The time sequence per se of these sleep substances may be responsible for the differential temporal changes in sleep. It is, therefore, assumed that a crucial order of multiple sleep substances may dynamically and differentially regulate sleep in the brain.

CT Medical Descriptors:
***sleep**
animal experiment
article
controlled study
drug effect
drug potentiation
intracerebroventricular drug administration
male
nonhuman
priority journal
rat
regulatory mechanism
sleep waking cycle
slow wave sleep
Drug Descriptors:
***muramyl dipeptide: IT, drug interaction**
***muramyl dipeptide: PD, pharmacology**
***uridine: IT, drug interaction**
***uridine: PD, pharmacology**

RN (uridine) **58-96-8**
CO Peptide institute (Japan)

L59 ANSWER 16 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 92246062 EMBASE
DN 1992246062
TI Effects of uridine in the treatment of diabetic neuropathy: An electrophysiological study.
AU Gallai V.; Mazzotta G.; Montesi S.; Sarchielli P.; Del Gatto F.
CS Department of Neurology, University of Perugia, Casella Postale 27,06100 Perugia, Italy
SO Acta Neurologica Scandinavica, (1992) 86/1 (3-7).
ISSN: 0001-6314 CODEN: ANRSAS
CY Denmark
DT Journal; Article
FS 003 Endocrinology
008 Neurology and Neurosurgery
030 Pharmacology
037 Drug Literature Index
LA English
SL English
AB The authors performed a controlled double-blind neurophysiological study (uridine vs placebo) in 40 diabetic patients with peripheral neuropathy. Twenty subjects were treated with uridine and 20 with placebo. The neurophysiological evaluation consisted of a study of the MCV of the median nerve, the common Peroneal, the posterior Tibial, the SCV of the radial nerve, the median and the sural as well as the amplitudes of the motor and sensory responses. The nerves examined were on the dominant side. The evaluations were preformed at baseline and after 60, 120, 180 days of therapy with a follow up control after 90 days from the completion of therapy. No statistically significant modifications were obsered in the placebo group. In the drug group, the neurophysiological parameters improved significantly from the 120th day post therapy compared with baseline and were maintained through to follow up. The authors discuss the results which demonstrated that treatment with uridine can bring about a neurophysiological improvement in peripheral nerves.
CT Medical Descriptors:

***diabetic neuropathy: DT, drug therapy**
adult
article
clinical article
female
human
male
nerve conduction
priority journal
Drug Descriptors:
***uridine: PD, pharmacology**
***uridine: DT, drug therapy**
RN (uridine) **58-96-8**

L59 ANSWER 17 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 91345012 EMBASE
DN 1991345012
TI Central depressant effects of N3-substituted 6-azauridines in mice.
AU Koshigami M.; Watanabe K.; Kimura T.; Yamamoto I.
CS Faculty of Pharmaceutical Sciences, Hokuriku University, 3-Ho Kanagawa-machi, Kanazawa 920-11, Japan
SO Chemical and Pharmaceutical Bulletin, (1991) 39/10 (2597-2599).
ISSN: 0009-2363 CODEN: CPBTAL
CY Japan
DT Journal; Article
FS 030 Pharmacology
037 Drug Literature Index
LA English
SL English
AB Central depressant effects in mice of N3-substituted 6-azauridines (6-AzUd) (1) were examined by intracerebroventricular (i.c.v.) injection. Eleven derivatives including alkyl-, benzyl-, xylyl- and phenylethyl-substitution onto the N3-position of 1 were synthesized and their pharmacological effects were evaluated using hypnotic activity, locomotor activity, motor incoordination and pentobarbital-induced sleep prolongation as indices. Six of 12 compounds showed the hypnotic activity. At a dose of 2 .mu.mol/mouse, the mean sleeping time induced by 1, N3-benzyl-6-AzUd (7), N3-o-xylyl-6-AzUd (8), N3-m-xylyl-6-AzUd (9), N3-p-xylyl-6-AzUd (10) and N3-.alpha.-phenylethyl-6-AzUd (11) was 14, 11, 45, 12, 9 and 16 min, respectively. These derivatives and N3-.beta.-phenylethyl-6-AzUd (12) (1.5 .mu.mol/mouse) significantly prolonged pentobarbital-induced (40 mg/kg, i.p.) sleeping time, whereas none of the N3-alkylated derivatives (methyl-, ethyl-, n-propyl-, n-butyl- and allyl-substitution) exerted the hypnotic activity or pentobarbital-induced sleep prolongation. Nucleoside 1 and its xylyl-derivatives (1.5 .mu.mol/mouse) significantly decreased locomotor activity of mice, their effects paralleled the hypnotic activity. These compounds (1.5 .mu.mol/mouse) also produced motor incoordination and potentiated the effect of diazepam-induced motor incoordination. These results indicate that 1 and its benzyl-related derivatives, but not alkyl-derivatives have a depressant effect on the central nervous system.
CT Medical Descriptors:
***hypnosis**
*locomotion
***sleep time**
animal experiment
article
controlled study
drug synthesis
intracerebroventricular drug administration
intraperitoneal drug administration
male
mouse
nonhuman
Drug Descriptors:
***azauridine: IT, drug interaction**

***azauridine: CB, drug combination**
*azauridine: AN, drug analysis
*azauridine: DV, drug development
***azauridine: CM, drug comparison**
***azauridine derivative: CM, drug comparison**
*azauridine derivative: DV, drug development
*azauridine derivative: AN, drug analysis
***azauridine derivative: CB, drug combination**
***azauridine derivative: IT, drug interaction**
***uridine derivative: IT, drug interaction**
***uridine derivative: CM, drug comparison**
***uridine derivative: DV, drug development**
***uridine derivative: AN, drug analysis**
***uridine derivative: CB, drug combination**
diazepam: IT, drug interaction
diazepam: CM, drug comparison
diazepam: CB, drug combination
pentobarbital: IT, drug interaction
pentobarbital: CM, drug comparison
pentobarbital: CB, drug combination
RN (azauridine) 54-25-1; (diazepam) 439-14-5; (pentobarbital) 57-33-0, 76-74-4
CO Aldrich; Yamanouchi pharmaceutical

L59 ANSWER 18 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 90236188 EMBASE
DN 1990236188
TI Memory deficits of aged male rats can be improved by pyrimidine nucleosides and n-acetyl-glutamine.
AU Drago F.; D'Agata V.; Valerio C.; Spadaro F.; Raffaele R.; Nardo L.; Grassi M.; Freni V.
CS Institute of Pharmacology, University of Catania Medical School, Viale A. Doria 6, 95125 Catania, Italy
SO Clinical Neuropharmacology, (1990) 13/4 (290-296).
ISSN: 0362-5664 CODEN: CLNEDB
CY United States
DT Journal; Article
FS 008 Neurology and Neurosurgery
020 Gerontology and Geriatrics
030 Pharmacology
037 Drug Literature Index
LA English
SL English
AB The pyrimidine nucleosides uridine (URI) and cytidine (CYT), alone or associated with n-acetyl-glutamine (NAG), were injected acutely or subchronically to aged (26 months old) male rats of the Sprague-Dawley strain. Learning and memory abilities of the animals were studied with tests of avoidance behavior. The acquisition of active avoidance behavior was studied with the shuttle-box test. A step-through type of passive avoidance task was used to examine the retention of passive avoidance responses. The acquisition of the active avoidance behavior and the retention of the passive avoidance response were reduced in aged animals as compared with those of young animals. Neither the acute treatment of old rats with URI and CYT alone nor that associated with NAG exerted any effect on the behavioral tests. In contrast, the subchronic treatment with URI and CYT was followed by a facilitation of acquisition of active avoidance behavior in the shuttle box and of retention of passive avoidance responses in the dark box. A more potent effect on the acquisition of the shuttle-box behavior and on the retention of passive avoidance reaction was found in animals treated subchronically with the pyrimidine nucleosides associated with NAG. These effects may be related to the role of pyrimidines in the synthesis of ribonucleic acid, which is indispensable for learning and memory processes.
CT Medical Descriptors:
***memory**
aged

rat
animal experiment
nonhuman
male
intraperitoneal drug administration
article
priority journal
Drug Descriptors:
***cytokine: PD, pharmacology**
***aceglutamide: PD, pharmacology**
***uridine: PD, pharmacology**
RN (aceglutamide) 2490-97-3; (uridine) **58-96-8**

L59 ANSWER 19 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 89176684 EMBASE
DN 1989176684
TI Effects of chronic treatment with uridine on striatal dopamine release and dopamine related behaviours in the absence or the presence of chronic treatment with haloperidol.
AU Agnati L.F.; Fuxe K.; Ruggeri M.; Pich E.M.; Benfenati F.; Volterra V.; Ungerstedt U.; Zini I.
CS Institute of Human Physiology, University of Modena, 41100 Modena, Italy
SO Neurochemistry International, (1989) 15/1 (107-113).
ISSN: 0197-0186 CODEN: NEUIDS
CY United Kingdom
DT Journal
FS 002 Physiology
030 Pharmacology
008 Neurology and Neurosurgery
037 Drug Literature Index
LA English
SL English
AB Uridine (15 mg/kg/day, i.p.), haloperidol (1 mg/kg/day, i.p.), uridine (15 mg/kg/day, i.p.) plus haloperidol (1 mg/kg/day, i.p.) or saline have been chronically administered to Sprague-Dawley male rats. Following 1 week of wash-out, the effects of these treatments on basal striatal dopamine (DA) release as well as on the DA release induced by an acute haloperidol challenge (2 mg/kg, i.p.) were studied by means of intracerebral microdialysis. Behavioural tests such as haloperidol-induced catalepsy or apomorphine-induced stereotypies were also performed 4-7 days after drug withdrawal. The chronic treatment with uridine alone or associated with haloperidol markedly reduced DA release induced by an acute haloperidol challenge. The behavioural studies also indicated a change in DA-related behaviours in these conditions. The animals chronically treated with uridine showed significant increases in the stereotypy scores and in the catalepsy induced by an acute haloperidol challange with respect to saline treated rats. The present results indicate that a chronic uridine treatment is able to potentiate the reduction of the striatal DA transmission induced by acute and chronic haloperidol treatment. This finding suggests the possibility to reduce the neuroleptic dose in the treatment of schizophrenia by combining neuroleptic and uridine treatments, thus making it possible to relieve some of the side effects of neuroleptic therapy.
CT Medical Descriptors:
***behavior**
***corpus striatum**
***dopamine release**
rat
schizophrenia
psychological aspect
nonhuman
animal experiment
male
intraperitoneal drug administration
priority journal
Drug Descriptors:

***haloperidol: PD, pharmacology**
***haloperidol: CB, drug combination**
***uridine: PD, pharmacology**
***uridine: CB, drug combination**
RN (haloperidol) 52-86-8; (uridine) **58-96-8**
CN (1) Serenase
CO (1) Istituto lusofarmaco (Italy)

L59 ANSWER 20 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 89027832 EMBASE
DN 1989027832
TI Effects of chronic uridine treatment on regional neuropeptide and tyrosine hydroxylase-like immunoreactivities in the brain of 12 month-old male rats.
AU Zoli M.; Agnati L.F.; Fuxe K.; Cintra A.; Grimaldi R.; Vanderhaeghen J.J.; Eneroth P.; Goldstein M.
CS Institute of Human Physiology, University of Modena, 41100 Modena, Italy
SO Neurochemistry International, (1988) 13/4 (499-508).
ISSN: 0197-0186 CODEN: NEUIDS
CY United Kingdom
DT Journal
FS 001 Anatomy, Anthropology, Embryology and Histology
003 Endocrinology
037 Drug Literature Index
LA English
SL English
AB Uridine was administered in the drinking water (0.5 mg/ml) in adult 6 month-old rats for 6 months. The mean daily dose of uridine was 12.5 mg/rat. The effects of this treatment on tyrosine hydroxylase, galanin, somatostatin, neuropeptide Y and cholecystokinin-like immunoreactivities were studied by means of semiquantitative immunocytchemistry using the peroxidase-antiperixodase procedure in combination with image analysis. A decrease of somatostatin, cholecystokinin and galanin-like immunoreactivities in nerve terminals was observed in various brain areas of 12 month-old animals compared with 3 month-old animals, while the levels of tyrosine hydroxylase-like immunoreactivity were unchanged. Uridine-treated animals showed a decrease of galanin, neuropeptide Y and cholecystokinin-like immunoreactivities in nerve terminals of some diencephalic areas and an increase of cholecystokinin-like immunoreactivity in nerve terminals of most of the telencephalic brain areas in comparison with vehicle treated animals of the same age. It is suggested that the pyrimidine nucleoside uridine can affect the synthesis and/or degradation of mRNAs involved in the synthesis of neuropeptide via direct nuclear actions and/or indirect actions involving effects on receptor activated phosphoinositide metabolism. Uridine offers a new way to modulate central peptide synapses.
CT Medical Descriptors:
***brain region**
cytochemistry
rat
animal experiment
animal cell
nonhuman
male
oral drug administration
priority journal
Drug Descriptors:
*neuropeptide
*tyrosine 3 monooxygenase
messenger rna
***uridine: PD, pharmacology**
RN (tyrosine 3 monooxygenase) 9036-22-0; (uridine) **58-96-8**

L59 ANSWER 21 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 88147812 EMBASE
DN 1988147812

TI Chronic uridine treatment reduces the level of [3H]spiperone-labelled dopamine receptors and enhances their turnover rate in striatum of young rats: Relationship to dopamine-dependent behaviours.
AU Farabegoli C.; Pich E.M.; Cimino M.; Agnati L.F.; Fuxe K.
CS Institute of Human Physiology, University of Modena, Modena, Italy
SO Acta Physiologica Scandinavica, (1988) 132/2 (209-216).
ISSN: 0001-6772 CODEN: APSCAX
CY Sweden
DT Journal
FS 002 Physiology
008 Neurology and Neurosurgery
030 Pharmacology
037 Drug Literature Index
LA English
SL English
AB The effect was studied of chronic uridine treatment on the recovery of striatal D-2 dopamine (DA) receptors after their irreversible blockade by N-ethoxycarbonyl-2-ethoxy-1,2-dihydroquinoline (EEDQ) in young (40 days old) and adult (14 months old) male rats using [3H]spiperone as radioligand. Chronic uridine treatment (15 mg kg-1 day-1, i.p., 14 days) causes a reduction of [3H]spiperone binding sites in striatum of young rats. This treatment also produces an increase in the rate of recovery of striatal [3H]spiperone-labelled DA receptors in young, but not in adult rats. Catalepsy and exploratory locomotor activity, two behaviours associated with blockade versus activation of DA receptors, were evaluated in the same rats. The behavioural recovery from the EEDQ-induced syndrome is more rapid in the young rats treated with uridine than in the saline-treated group. The behavioural recovery in old rats was not affected by chronic uridine treatment. Thus, in young rats the pyrimidine nucleoside uridine may modulate the steady state and the turnover rate of striatal D-2 DA receptors.
CT Medical Descriptors:
***catalepsy**
***corpus striatum**
*locomotion
age
rat
priority journal
animal experiment
animal cell
nonhuman
male
intraperitoneal drug administration
Drug Descriptors:
***dopamine receptor**
***2 ethoxy 1(2h) quinolinecarboxylic acid ethyl ester: PD, pharmacology**
***spiperone: PD, pharmacology**
***uridine: PD, pharmacology**
RN (2 ethoxy 1(2h) quinolinecarboxylic acid ethyl ester) 16357-59-8; (spiperone) 749-02-0; (uridine) **58-96-8**
CO Sigma (United States)

L59 ANSWER 22 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 87234969 EMBASE
DN 1987234969
TI N-substituted oxopyrimidines and nucleosides: Structure-activity relationship for hypnotic activity as central nervous system depressant.
AU Yamamoto I.; Kimura T.; Tateoka Y.; Watanabe K.; Ho I.K.
CS Department of Hygienic Chemistry, School of Pharmacy, Hokuriku University, Kanazawa 920-11, Japan
SO Journal of Medicinal Chemistry, (1987) 30/12 (2227-2231).
ISSN: 0022-2623 CODEN: JMCMAR
CY United States
DT Journal
FS 008 Neurology and Neurosurgery

030 Pharmacology
037 Drug Literature Index
LA English
AB N3-Benzyluridine (3-(phenylmethyl)-1-.beta.-D-ribofuranosyluracil) (1f) and its related compounds were synthesized and evaluated for hypnotic activity as central depressants. The primary structural modification has been carried out at the N3 position of the pyrimidine ring in uridine. N3-Benzyl-substituted uridine exhibited hypnotic activity as well as pentobarbital (PB) induced sleep effect on mice when administered by intracerebroventricular (icv) injection. From this result, the secondary modification was performed, namely, converting the benzyl group into a benzyl analogous group. These compounds also showed hypnotic activity, but their intensities were varied. Thirdly, changing the sugar moiety was investigated; however, it was found to be necessary for hypnotic activity. In general, introduction of benzyl analogous groups at the N3 position of uridine increased the hypnotic activity, and modification of the sugar moiety decreased the activity. Intravenous (iv) administration failed to indicate hypnotic activity in most of the compounds tested. However, modified sugars such as 2',3',5'-tri-O-methyl or -acetyl derivatives of 1f elicited hypnotic activity by iv injection. The majority of compounds were found to show potentiation of the PB-induced sleep, and their effects were in parallel with the hypnotic activity. The result clearly indicates that the benzyl group and .beta.-D-ribofuranosyl, at the N3 and N1 positions, respectively, are necessary for hypnotic activity. The critical portion of the chemical structure for both effects appears to be the uridine moiety.
CT Medical Descriptors:
***central nervous system depression**
***hypnosis**
***sleep**
structure activity relation
drug analysis
drug comparison
animal experiment
nonhuman
mouse
central nervous system
Drug Descriptors:
*3 benzyluridine: AN, drug analysis
*3 benzyluridine: DV, drug development
***3 benzyluridine: PD, pharmacology**
***3 benzyluridine: CM, drug comparison**
***uridine derivative: AN, drug analysis**
***uridine derivative: DV, drug development**
***uridine derivative: PD, pharmacology**
***uridine derivative: CM, drug comparison**
tegafur
uracil
unclassified drug
RN (tegafur) 17902-23-7; (uracil) 66-22-8

L59 ANSWER 23 OF 23 EMBASE COPYRIGHT 2000 ELSEVIER SCI. B.V.
AN 87233360 EMBASE
DN 1987233360
TI Hypnotic activity of N3-benzylthymidine on mice.
AU Yamamoto I.; Kimura T.; Tateoka Y.; Watanabe K.; Ho I.K.
CS Department of Hygienic Chemistry, School of Pharmacy, Hokuriku University, Kanazawa 920-11, Japan
SO Life Sciences, (1987) 41/26 (2791-2797).
ISSN: 0024-3205 CODEN: LIFSAK
CY United States
DT Journal
FS 030 Pharmacology
037 Drug Literature Index
LA English
AB The pharmacological effect in mice of N3-benzylthymidine (N3-ByTd) was examined by two routes of administration; intravenous (i.v.) and

intracerebroventricular (i.c.v.), and compared with the effect of administration of N3-benzyluridine (N3-ByUd) previously reported. Hypnotic activity, pentobarbital (PB)-induced sleeping time, motor incoordination and spontaneous activity were used as indices of pharmacological effects. N3-ByTd (0.5-2.0 .mu.mol/mouse, i.c.v.) and N3-ByUd (1.5-3.0 .mu.mol/mouse, i.c.v.) were found to possess dose-dependent hypnotic activity, and N3-ByTd had more potent hypnotic activity than N3-ByUd. Both N3-ByTd and N3-ByUd (0.5 and 1.0 .mu.mol/mouse, i.c.v., respectively) showed a synergistic effect on PB-induced sleep, although their parent compounds, thymidine (Td) and uridine (Ud), did not potentiate the activity at each dose. In motor incoordination, the effect of N3-ByTd (0.5 .mu.mol/mouse) continued for 6 hr after i.c.v. injection. All compounds decreased the spontaneous activity of mice by i.c.v. administration. Furthermore, both N3-ByTd and N3-ByUd decreased the activity, when they were administered i.v. These results reveal that both N3-benzylpyrimidine nucleosides have more direct depressant effects on the central nervous system (CNS) than the parent compounds. Among the pyrimidine nucleoside derivatives tested, N3-ByTd was found to be the most potent hypnotic substance.

CT Medical Descriptors:
***central nervous system depression**
***sleep time**
drug comparison
preliminary communication
nonhuman
central nervous system
mouse
animal experiment
Drug Descriptors:
*hypnotic agent
***thymidine: PD, pharmacology**
***thymidine: CM, drug comparison**
***uridine: PD, pharmacology**
***uridine: CM, drug comparison**
RN (thymidine) 50-89-5; (uridine) **58-96-8**
CO Wako (Japan); Kohjin (Japan)

=> fil biosis

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FILE 'BIOSIS' ENTERED AT 08:34:27 ON 08 AUG 2000
L60 3730 S L1
L61 78 S L60 AND 0700?/CC
L62 586 S L60 AND 12100/CC
L63 68 S L60 AND 1750?/CC
L64 372 S L60 AND 2050?/CC
L65 61 S L60 AND 2100?/CC
L66 922 S L61-L65
L67 96 S L66 AND 12512/CC

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L68          358 S L66 AND 220?/CC
L69          372 S L67,L68
L70          368 S L69 AND PY<=1998
L71           43 S L70 AND 00520/CC
L72           64 S L70 AND (CONFERENCE? OR CONGRESS? OR POSTER? OR SYMPOS? OR ME
L73            4 S L70 AND MEET/SO
L74           64 S L71-L73
L75            8 S L74 AND (ORAL URIDINE OR URIDINE COADMINISTRATION OR SCLEROSI
L76           12 S L70 AND *12512/CC
L77           94 S L70 AND (*22005 OR *22024 OR *22026)/CC
L78           74 S L76,L77 NOT L74
L79           18 S L78 AND (LEARNING OR PSYCHOTROP? OR AVOIDANC? OR ROTATION OR
L80           26 S L75,L79

     FILE 'BIOSIS' ENTERED AT 08:52:37 ON 08 AUG 2000

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L80  ANSWER 1 OF 26  BIOSIS  COPYRIGHT 2000 BIOSIS
AN   1997:405298  BIOSIS
DN   PREV199799711501
TI   A longitudinal study of cognitive functioning in patients with
     classical galactosaemia, including a cohort treated with oral uridine.
AU   Manis, F. R. (1); Cohn, L. B.; McBride-Chang, C.; Wolff, J. A.; Kaufman,
     F. R.
CS   (1) Dep. Psychol., Univ. Southern California, Los Angeles, CA 90089-1061
     USA
SO   Journal of Inherited Metabolic Disease, (1997) Vol. 20, No. 4, pp.
     549-555.
     ISSN: 0141-8955.
DT   Article
LA   English
AB   Existing longitudinal data on patients with classical galactosaemia
     suggests that neurocognitive functioning is impaired and, in isolated case
     reports, may show a decline in performance over time. The present study
     explored whether there are long-term changes in cognitive abilities in
     patients with galactosaemia and whether oral uridine can improve
     neurocognitive performance. Thirty-five patients (18 males, 17 females),
     29 of whom received oral uridine powder at 150 mg/kg per day (divided
     dose, three times daily), were evaluated over a 2-5-year period with the
     Woodcock-Johnson Revised Cognitive Abilities Test, three academic
     achievement tests, and the Beery Test of Visual Motor Integration. Results
     showed that the uridine cohort and a comparison group that received only
     dietary restriction made small gains in cognitive performance over the
     treatment period and the size of the gains did not differ significantly.
     Seven subjects who started uridine prior to the age of 14 months did not
     differ significantly in their cognitive test scores at an average age of
     3.5 years from a group of older children who had begun treatment at 4.5
     years of age. These results provide no support for any developmental or
     uridine-treatmentrelated chance in cognitive functioning for this sample
     of galactosaemic subjects.
CC   Behavioral Biology - Human Behavior   07004
     Pathology, General and Miscellaneous - Therapy   12512
     Metabolism - Carbohydrates  *13004
     Metabolism - Metabolic Disorders  *13020
     Psychiatry - Psychophysiology  *21003
     Pharmacology - Drug Metabolism; Metabolic Stimulators  *22003
     Pharmacology - Clinical Pharmacology   *22005
BC   Hominidae  *86215
IT   Major Concepts
        Behavior; Metabolism; Pharmacology
IT   Chemicals & Biochemicals
        URIDINE
IT   Miscellaneous Descriptors
        BEHAVIOR; COGNITIVE FUNCTION; GALACTOSEMIA; GENETIC DISEASE; METABOLIC
```

DISEASE; METABOLIC-DRUG; METABOLISM; ORAL DOSAGE; PATIENT; PHARMACOLOGY; URIDINE
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates
RN **58-96-8** (URIDINE)

L80 ANSWER 2 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1996:466219 BIOSIS
DN PREV199699188575
TI Multi-infarct **dementia**: Modification of the P300 **cognitive** event-related potential in patients treated with the association of cytidine and uridine.
AU Gallai, V.; Alberti, A.; Mazzotta, G.
CS Clin. Neurol., Univ. degli Studi, Perugia Italy
SO Rivista di Neuropsichiatria e Scienze Affini, (1995) Vol. 41, No. 1, pp. 1-9.
ISSN: 0035-6352.
DT Article
LA Italian
SL Italian; English
AB In Italy as in all western countries the mean age of the population is increasing progressively with consequent increase of the degenerative pathologies of the central nervous system, making extremely important the question of the cognitive decline. Although the majority of the dementia syndromes are due to Alzheimer's disease and Alzheimer type, another important cause of dementia is Multi Infarct Dementia (MID), which is related to alterations of the cerebral blood flow. The present study was designed to evaluate the efficacy of Cytidine and Uridine in subjects with reduced mental capacity following to MID by means the event-related potential P300. The P300 is a neurophysiological method used to investigate cerebral electrical activity in the cognitive processing of information analysis. This potential was found to be altered in subjects affected by dementia. The present study was performed in 20 patients affected by multi-infarct dementia (MID) treated with Cytidine and Uridine. The patients, after a period of washout, were evaluated by electrophysiological examination performed at baseline and after 60 days. The event-related potential P300 was performed by an "odd-ball" paradigm with an acoustic modality; the patients were also assessed with the Digit Span, a sub-test of the Wechsler Adult Intelligence Scale to evaluate attention and short-term memory and with the Mini Mentale State. In the patients examined, the findings relevant to the study of the P300 showed a significant decrease in latency values compared to baseline. On the basis of this investigation it has been demonstrated that the variations in the registrations can be correlated to the improved neuronal activity following treatment with Cytidine and Uridine.
CC **Behavioral Biology - Human Behavior 07004**
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Pathology, General and Miscellaneous - Therapy *12512
Blood, Blood-Forming Organs and Body Fluids - Blood and Lymph Studies *15002
Nervous System - Physiology and Biochemistry *20504
Nervous System - Pathology *20506
Psychiatry - Psychopathology; Psychodynamics and Therapy *21002
Pharmacology - Clinical Pharmacology *22005
Pharmacology - Neuropharmacology *22024
BC Hominidae *86215
IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Nervous System (Neural Coordination); Neurology (Human Medicine, Medical Sciences); Pathology; Pharmacology; Psychiatry (Human Medicine, Medical Sciences)
IT Chemicals & Biochemicals
CYTIDINE; URIDINE

IT Miscellaneous Descriptors
AUTONOMIC-DRUG; BEHAVIORAL AND MENTAL DISORDERS; CEREBRAL BLOOD FLOW; CYTIDINE; MULTI-INFARCT DEMENTIA; NERVOUS SYSTEM DISEASE; NEUROLOGY; PATIENT; PHARMACOLOGY; P300 COGNITIVE EVENT-RELATED POTENTIAL; URIDINE
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates
RN 65-46-3 (CYTIDINE)
58-96-8 (URIDINE)

L80 ANSWER 3 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1996:26741 BIOSIS
DN PREV199698598876
TI Uridine reduces **rotation** induced by L-dopa and methamphetamine in 6-OHDA-treated rats.
AU Myers, Carol S.; Fisher, Hans; Wagner, George C. (1)
CS (1) Dep. Psychol., Busch Campus, Rutgers Univ., New Brunswick, NJ 08903 USA
SO Pharmacology Biochemistry and Behavior, (1995) Vol. 52, No. 4, pp. 749-753.
ISSN: 0091-3057.
DT Article
LA English
AB The pyrimidine nucleoside uridine may reduce side effects associated with antipsychotic medication by interacting with dopamine or GABA neurotransmission. Male Sprague-Dawley rats were used to investigate coadministration of uridine with agents that alter food intake (amphetamine, haloperidol, and chlordiazepoxide) and locomotor activity (methamphetamine and L-dopa). Results indicated that chronic uridine (32.0 mg/kg, intraperitoneally (IP)) alone did not alter milk intake or reduction of milk intake induced by amphetamine (dose range 0.5-2.0 mg/kg, IP) or haloperidol (0.125-1.0 mg/kg, IP), nor did it alter the biphasic response induced by chlordiazepoxide (5.0-40.0 mg/kg, IP). However, uridine-treated animals with unilateral striatal lesions exhibited no rotational behavior in the absence of drug challenge, but showed decreased rotation induced by the dopamine agonist, L-dopa (50.0-200.0 mg/kg, IP) compared with controls. In addition, uridine-treated rats exhibited reduced rotation after repeated injections of methamphetamine (4.0 mg/kg, IP) in contrast to increasingly greater rotation observed in control animals. These results are further evidence that chronic uridine may alter drug-induced dopaminergic activity without exerting effects itself.
CC **Behavioral Biology - Animal Behavior *07003**
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Nervous System - Pathology *20506
Psychiatry - Psychopathology; Psychodynamics and Therapy *21002
Pharmacology - Neuropharmacology *22024
Pharmacology - Psychopharmacology *22026
BC Muridae *86375
IT Major Concepts
Behavior; Nervous System (Neural Coordination); Pharmacology
IT Chemicals & Biochemicals
URIDINE; L-DOPA; METHAMPHETAMINE
IT Miscellaneous Descriptors
ANTIPSYCHOTIC AGENT; PSYCHOSIS; URIDINE
ORGN Super Taxa
Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
Muridae (Muridae)
ORGN Organism Superterms
animals; chordates; mammals; nonhuman vertebrates; nonhuman mammals; rodents; vertebrates
RN **58-96-8** (URIDINE)
59-92-7 (L-DOPA)

537-46-2 (METHAMPHETAMINE)

L80 ANSWER 4 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1995:426838 BIOSIS
DN PREV199598441138
TI Neurochemical and behavioral effects of haloperidol and risperidone: Evaluation of **uridine coadministration** with **antipsychotic** drugs.
AU Myers, C. S.; Michna, L.; Aknay, N.; Fisher, H.; Wagner, G. C.
CS Dep. Psychol., Rutgers Univ., New Brunswick, NJ 08903 USA
SO **Society for Neuroscience Abstracts**, (1995) Vol. 21, No. 1-3, pp. 195.
Meeting Info.: **25th Annual Meeting of the Society for Neuroscience** San Diego, California, USA November 11-16, 1995
ISSN: 0190-5295.
DT Conference
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Behavioral Biology - Animal Behavior *07003
Behavioral Biology - Conditioning *07005
Biochemical Studies - General 10060
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines *10062
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Metabolism - Proteins, Peptides and Amino Acids *13012
Endocrine System - Neuroendocrinology *17020
Nervous System - Physiology and Biochemistry *20504
Pharmacology - Neuropharmacology *22024
Pharmacology - Psychopharmacology *22026
Toxicology - Pharmacological Toxicology *22504
BC Muridae *86375
IT Major Concepts
Behavior; Biochemistry and Molecular Biophysics; Endocrine System (Chemical Coordination and Homeostasis); Metabolism; Nervous System (Neural Coordination); Pharmacology; Toxicology
IT Chemicals & Biochemicals
HALOPERIDOL; RISPERIDONE; URIDINE; 5-HYDROXYTRYPTAMINE
IT Miscellaneous Descriptors
ANTIPSYCHOTIC-DRUG; CONDITIONED AVOIDANCE RESPONSE DISRUPTION; EXTRAPYRAMIDAL SIDE EFFECTS; HALOPERIDOL; **MEETING ABSTRACT**; **MEETING POSTER**; RISPERIDONE; 5-HYDROXYTRYPTAMINE TURNOVER
ORGN Super Taxa
Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
rat (Muridae)
ORGN Organism Superterms
animals; chordates; mammals; nonhuman mammals; nonhuman vertebrates; rodents; vertebrates
RN 52-86-8 (HALOPERIDOL)
106266-06-2 (RISPERIDONE)
58-96-8 (URIDINE)
50-67-9 (5-HYDROXYTRYPTAMINE)

L80 ANSWER 5 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1995:237308 BIOSIS
DN PREV199598251608
TI Longitudinal neurocognitive data on patients (PTS) with classical galactosemia (G), including a cohort treated with **oral uridine**.
AU Manis, Frank R. (1); Cohn, Linda; McBride-Chang, Catherine; Wolff, Jon A.; Kaufman, Francine R.
CS (1) Dep. Psychol., USC Sch. Med., Los Angeles, CA USA
SO Pediatric Research, (1994) Vol. 37, No. 4 PART 2, pp. 150A.
Meeting Info.: **105th Annual Meeting of the American Pediatric Society and the 64th Annual Meeting of the Society for Pediatric Research** San Diego, California, USA May 7-11, 1995

ISSN: 0031-3998.
DT Conference
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Genetics and Cytogenetics - Human *03508
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Carbohydrates 10068
Enzymes - Physiological Studies *10808
Pathology, General and Miscellaneous - Therapy *12512
Metabolism - Carbohydrates *13004
Metabolism - Metabolic Disorders *13020
Nervous System - Pathology *20506
Psychiatry - Psychopathology; Psychodynamics and Therapy *21002
Psychiatry - Mental Retardation *21006
Pharmacology - Drug Metabolism; Metabolic Stimulators *22003
Pediatrics *25000
BC Hominidae *86215
IT Major Concepts
Enzymology (Biochemistry and Molecular Biophysics); Genetics; Metabolism; Neurology (Human Medicine, Medical Sciences); Pathology; Pediatrics (Human Medicine, Medical Sciences); Pharmacology; Psychiatry (Human Medicine, Medical Sciences)
IT Chemicals & Biochemicals
URIDINE
IT Miscellaneous Descriptors
IMPAIRED NEUROCOGNITIVE FUNCTIONING; INBORN METABOLIC ERROR; **MEETING ABSTRACT; MEETING POSTER;** MENTAL RETARDATION; METABOLIC-DRUG; PEDIATRICS; URIDINE
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates
RN **58-96-8** (URIDINE)

L80 ANSWER 6 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1995:237301 BIOSIS
DN PREV199598251601
TI A 5 year study evaluation the use of **oral uridine** in patients (PTS) with classical galactosemia (G.
AU Kaufman, Francine R. (1); Xu, Yan-Kang; Ng, Won Gin; Nelson, Marvin D.; Gray, Susan; Wolff, Jon A.
CS (1) Dep. Pediatrics, USC Sch. Med., Los Angeles, CA USA
SO Pediatric Research, (1994) Vol. 37, No. 4 PART 2, pp. 149A.
Meeting Info.: **105th Annual Meeting of the American Pediatric Society and the 64th Annual Meeting of the Society for Pediatric Research** San Diego, California, USA May 7-11, 1995
ISSN: 0031-3998.
DT Conference
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Genetics and Cytogenetics - Human *03508
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Carbohydrates 10068
Enzymes - Physiological Studies *10808
Pathology, General and Miscellaneous - Therapy *12512
Metabolism - Carbohydrates *13004
Metabolism - Metabolic Disorders *13020
Reproductive System - Physiology and Biochemistry *16504
Dental and Oral Biology - General; Methods 19001
Nervous System - Physiology and Biochemistry *20504

Pharmacology - Drug Metabolism; Metabolic Stimulators *22003
Pharmacology - Clinical Pharmacology *22005
Pharmacology - Neuropharmacology *22024
Pharmacology - Reproductive System; Implantation Studies *22028
Pediatrics *25000
BC Hominidae *86215
IT Major Concepts
Enzymology (Biochemistry and Molecular Biophysics); Genetics; Metabolism; Nervous System (Neural Coordination); Pathology; Pediatrics (Human Medicine, Medical Sciences); Pharmacology; Reproductive System (Reproduction)
IT Chemicals & Biochemicals
URIDINE; URIDINE DIPHOSPHATE GALACTOSE
IT Miscellaneous Descriptors
INBORN METABOLIC ERROR; **MEETING ABSTRACT; MEETING POSTER**; METABOLIC-DRUG; NEUROLOGIC FUNCTION; OVARIAN FUNCTION; PEDIATRICS; URIDINE; URIDINE DIPHOSPHATE GALACTOSE
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates
RN **58-96-8** (URIDINE)
2956-16-3 (URIDINE DIPHOSPHATE GALACTOSE)

L80 ANSWER 7 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1994:512986 BIOSIS
DN PREV199497525986
TI Effect of uridine on disruption of conditioned avoidance responding induced by traditional and atypical **antipsychotics**.
AU Myers, C. S.; Fisher, H.; Wagner, G. C.
CS Dep. Psychol., Rutgers Univ., New Brunswick, NJ 08903 USA
SO **Society for Neuroscience Abstracts**, (1994) Vol. 20, No. 1-2, pp. 825.
Meeting Info.: **24th Annual Meeting of the Society for Neuroscience** Miami Beach, Florida, USA November 13-18, 1994
ISSN: 0190-5295.
DT Conference
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Behavioral Biology - Animal Behavior *07003
Behavioral Biology - Conditioning *07005
Biochemical Studies - General 10060
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Metabolism - Nucleic Acids, Purines and Pyrimidines *13014
Nervous System - Physiology and Biochemistry *20504
Pharmacology - Neuropharmacology *22024
Pharmacology - Psychopharmacology *22026
BC Muridae *86375
IT Major Concepts
Behavior; Metabolism; Nervous System (Neural Coordination); Pharmacology
IT Chemicals & Biochemicals
URIDINE
IT Miscellaneous Descriptors
BEHAVIOR; CATECHOLAMINES; **MEETING ABSTRACT; MEETING POSTER**; MONOAMINES
ORGN Super Taxa
Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
rat (Muridae)
ORGN Organism Superterms
animals; chordates; mammals; nonhuman mammals; nonhuman vertebrates; rodents; vertebrates

RN **58-96-8** (URIDINE)

L80 ANSWER 8 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1994:413648 BIOSIS
DN PREV199497426648
TI Anti-human immunodeficiency virus type 1 therapy and peripheral **neuropathy**: Prevention of 2',3'-dideoxycytidine toxicity in PC12 cells, a neuronal model, by uridine and pyruvate.
AU Keilbaugh, Sue A.; Hobbs, Gregory A.; Simpson, Melvin V. (1)
CS (1) Dep. Biochem. Cell Biol., State Univ. New York, Stony Brook, NY 11794-5215 USA
SO Molecular Pharmacology, (1993) Vol. 44, No. 4, pp. 702-706. ISSN: 0026-895X.
DT Article
LA English
AB A strategy for preventing or delaying the peripheral neuropathy induced by 2',3'-dideoxycytidine (ddC) therapy in patients with acquired immunodeficiency syndrome was suggested by findings, in two laboratories, that cultured avian and mammalian cells devoid of mitochondrial DNA continue to replicate at virtually normal rates, provided that the medium is supplemented with uridine and pyruvate. Inasmuch as it is likely that a depletion of mitochondrial DNA also takes place in neuronal cells exposed to ddC, we used PC12 cells, the neuronal model we have reported on previously, in an attempt to rescue these cells from the deleterious effects of ddC. We first show, using undifferentiated PC12 cells, that DNA replication is impaired in mitochondria isolated from cells grown in the presence of ddC. Then, using growth rate as a criterion of the well-being of the cells, we show that the addition of uridine and pyruvate to uninduced cells growing in the presence of ddC results in an average rescue efficiency of 51%, based on the uridine/pyruvate-treated control. This value increases considerably at substantially higher concentrations of uridine alone. Rescue efficiencies of differentiated cells, which do not proliferate, were assessed using neurite outgrowth and neurite survival as criteria. Here the rescue efficiency is 56%, based on the uridine/pyruvate-treated control. In addition, uridine and pyruvate prolong the viability of ddC-treated cells and maintain their healthy appearance; without these compounds, the ddC-treated cells have an abnormal morphology and die off quite rapidly.
CC Cytology and Cytochemistry - Animal *02506
Biochemical Studies - General 10060
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Pathology, General and Miscellaneous - Therapy *12512
Nervous System - Pathology *20506
Pharmacology - Clinical Pharmacology 22005
Toxicology - Pharmacological Toxicology *22504
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
Medical and Clinical Microbiology - Virology *36006
Chemotherapy - Antiviral Agents *38506
BC Retroviridae 02623
Hominidae *86215
IT Major Concepts
Cell Biology; Clinical Immunology (Human Medicine, Medical Sciences); Infection; Neurology (Human Medicine, Medical Sciences); Pathology; Pharmacology; Toxicology
IT Chemicals & Biochemicals
2',3'-DIDEOXYCYTIDINE; URIDINE; PYRUVATE
IT Miscellaneous Descriptors
ACQUIRED IMMUNODEFICIENCY SYNDROME; ANTIVIRAL AGENT; MITOCHONDRIAL DNA; 2,3-DIDEOXYCYTIDINE
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia; Retroviridae: Viruses
ORGN Organism Name
Hominidae (Hominidae); Retroviridae (Retroviridae)
ORGN Organism Superterms

animals; chordates; humans; mammals; microorganisms; primates; vertebrates; viruses
RN 7481-89-2 (2',3'-DIDEOXYCYTIDINE)
58-96-8 (URIDINE)
57-60-3 (PYRUVATE)

L80 ANSWER 9 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1994:413626 BIOSIS
DN PREV199497426626
TI Uridine potentiates haloperidol's disruption of conditioned **avoidance** responding.
AU Myers, Carol S.; Fisher, Hans; Wagner, George C. (1)
CS (1) Dep. Psychol., Rutgers Univ., New Brunswick, NJ 08903 USA
SO Brain Research, (1994) Vol. 651, No. 1-2, pp. 194-198.
ISSN: 0006-8993.
DT Article
LA English
AB The pyrimidine nucleoside, uridine, has been proposed as a potential supplement in the treatment of psychosis based on its ability.to reduce haloperidol-induced dopamine release. These experiments investigated the effect of uridine (32 mg/kg, i.p.) coadministered with the neuroleptic haloperidol, on rats engaged in one way conditioned avoidance responding. Uridine itself had no effect on animals' performance, while haloperidol (dose range 0.05-0.4 mg/kg, i.p., 90 min before test session) decreased number of avoidances and increased avoidance and escape latencies in a dose-dependent manner. When coadministered with haloperidol, uridine significantly potentiated the disruption of avoidance and avoidance latency induced by haloperidol. This potentiation was still evident after chronic (27 days) uridine treatment. Importantly, coadministration of uridine did not potentiate haloperidol-induced increase of escape latency. The potentiation of haloperidol-induced disruption of conditioned avoidance responding suggests that uridine coadministration might enhance the antipsychotic action of traditional neuroleptics. This would allow for a reduction in the therapeutic dose of the antipsychotic, thereby reducing side effect frequency.
CC **Behavioral Biology - Animal Behavior *07003**
Behavioral Biology - Conditioning 07005
Biochemical Studies - General 10060
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Movement *12100
Pathology, General and Miscellaneous - Therapy 12512
Nervous System - Pathology *20506
Pharmacology - Psychopharmacology *22026
Toxicology - Pharmacological Toxicology *22504
BC Muridae *86375
IT Major Concepts
Behavior; Nervous System (Neural Coordination); Pharmacology; Physiology; Toxicology
IT Chemicals & Biochemicals
URIDINE; HALOPERIDOL
IT Miscellaneous Descriptors
ANTIPSYCHOTIC; DYSKINESIA; HALOPERIDOL; PHARMACEUTICAL ADJUNCT; PSYCHOSIS; TOXICITY; URIDINE
ORGN Super Taxa
Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
rat (Muridae)
ORGN Organism Superterms
animals; chordates; mammals; nonhuman mammals; nonhuman vertebrates; rodents; vertebrates
RN **58-96-8** (URIDINE)
52-86-8 (HALOPERIDOL)

L80 ANSWER 10 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1994:5041 BIOSIS
DN PREV199497018041

TI Protective effects of uridine on methamphetamine-induced **dopamine depletions**.
AU Myers, C. S.; Wagner, G. C.
CS Dep. Psychol., Rutgers Univ., New Brunswick, NJ 08903 USA
SO **Society for Neuroscience Abstracts**, (1993) Vol. 19, No. 1-3, pp. 405.
Meeting Info.: **23rd Annual Meeting of the Society for Neuroscience** Washington, D.C., USA November 7-12, 1993
ISSN: 0190-5295.
DT Conference
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Endocrine System - Neuroendocrinology *17020
Nervous System - Physiology and Biochemistry *20504
Nervous System - Pathology *20506
Pharmacology - Neuropharmacology *22024
Toxicology - Antidotes and Preventative Toxicology *22505
BC Muridae *86375
IT Major Concepts
Endocrine System (Chemical Coordination and Homeostasis); Nervous System (Neural Coordination); Pharmacology; Toxicology
IT Chemicals & Biochemicals
URIDINE; METHAMPHETAMINE; DOPAMINE
IT Miscellaneous Descriptors
MEETING ABSTRACT; MEETING POSTER
ORGN Super Taxa
Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
rat (Muridae)
ORGN Organism Superterms
animals; chordates; mammals; nonhuman mammals; nonhuman vertebrates; rodents; vertebrates
RN **58-96-8** (URIDINE)
537-46-2 (METHAMPHETAMINE)
51-61-6 (DOPAMINE)

L80 ANSWER 11 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1992:480655 BIOSIS
DN BA94:112030
TI EFFECTS OF URIDINE IN THE TREATMENT OF **DIABETIC NEUROPATHY** AN ELECTROPHYSIOLOGICAL STUDY.
AU GALLAI V; MAZZOTTA G; MONTESI S; SARCHIELLI P; DEL GATTO F
CS UNIV. PERUGIA, CASELLA POSTALE 27, 06100 PERUGIA SUCC. 3, ITALY.
SO ACTA NEUROL SCAND, (1992) 86 (1), 3-7.
CODEN: ANRSAS. ISSN: 0001-6314.
FS BA; OLD
LA English
AB The authors performed a controlled double-blind neurophysiological study (uridine vs placebo) in 40 diabetic patients with peripheral neuropathy. Twenty subjects were treated with uridine and 20 with placebo. The neurophysiological evaluation consisted of a study of the MCV of the median nerve, the common Peroneal, the **posterior** Tibial, the SCV of the radial nerve, the median and the sural as well as the amplitudes of the motor and sensory responses. The nerves examined were on the dominant side. The evaluations were performed at baseline and after 60, 2120, 180 days of therapy with a follow up control after 90 days from the completion of therapy. No statistically significant modifications were observed in the placebo group. In the drug group, the neurophysiological parameters improved significantly from the 120th day post therapy compared with baseline and were maintained through to follow up. The authors discuss the results which demonstrated that treatment with uridine can bring about a neurophysiological improvement in peripheral nerves.
CC Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062

Biochemical Studies - Carbohydrates 10068
Pathology, General and Miscellaneous - Therapy *12512
Metabolism - Carbohydrates *13004
Metabolism - Metabolic Disorders *13020
Endocrine System - Pancreas *17008
Muscle - Pathology *17506
Sense Organs, Associated Structures and Functions - Pathology *20006
Nervous System - Pathology *20506
Pharmacology - Drug Metabolism; Metabolic Stimulators *22003
Pharmacology - Clinical Pharmacology *22005
BC Hominidae 86215
IT Miscellaneous Descriptors
HUMAN METABOLIC-DRUG MOTOR CONDUCTION SENSORY CONDUCTION PERIPHERAL NEUROPATHY
RN **58-96-8** (URIDINE)

L80 ANSWER 12 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1991:369905 BIOSIS
DN BA92:58130
TI PHARMACOLOGICAL ANALYSIS OF THE **ANXIOLYTIC** ACTIVITY OF URIDINE.
AU KARKISHCHENKO N N; KHAITIN M I; SIMKINA YU N
CS LAB. MOL. PHARMACOL., RES. INST. PHYS. ORG. CHEM., ROSTOV UNIV., ROSTOV-NA-DONU 344104, USSR.
SO FARMAKOL TOKSIKOL (MOSC), (1991) 54 (1), 16-18.
CODEN: FATOAO. ISSN: 0014-8318.
FS BA; OLD
LA Russian
AB The effects of prazosin (0.6 and 3 mg/kg), propranolol (5 mg/kg), haloperidol (0.1 and 0.5 mg/kg) and ciproheptaline (0.3 and 0.6 mg/kg) on the anxiolytic (anticonflict) action of a pyrimidine ribonucleoside uridine, a hypothetic endogenic regulator of anxiety states were studied in the experiments on male CBWA mice. It was found that the degree of the anxiolytic effect of uridine decreases at the blockade of .alpha.1, .beta.2, D2 and H-receptors and significantly increases at the blockade of 5-HT2-receptors. This suggests the involvement of the mentioned receptors in the processes of realization of uridine anxiolytic activity as well as the presence of the central serotonin-negative component in the mechanisms of action of uridine.
CC Cytology and Cytochemistry - Animal *02506
Behavioral Biology - Animal Behavior *07003
Biochemical Studies - General 10060
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biophysics - Membrane Phenomena *10508
Physiology, General and Miscellaneous - Methods *12006
Nervous System - Physiology and Biochemistry *20504
Psychiatry - Psychopathology; Psychodynamics and Therapy *21002
Psychiatry - Psychophysiology *21003
Pharmacology - Neuropharmacology *22024
Pharmacology - Psychopharmacology *22026
BC Muridae 86375
IT Miscellaneous Descriptors
MOUSE PHARMACOLOGICAL TOOLS ANXIETY ENDOGENOUS REGULATION ALPHA-1-ADRENOCEPTOR BETA-2-ADRENOCEPTOR DOPAMINE D2 RECEPTOR HISTAMINE H1 RECEPTOR 5 HYDROXYTRYPTAMINE-2 RECEPTOR SEROTONIN PHARMACOTHERAPEUTIC RELEVANCE
RN 50-67-9 (SEROTONIN)
51-45-6 (HISTAMINE)
51-61-6 (DOPAMINE)
58-96-8 (URIDINE)

L80 ANSWER 13 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1991:80007 BIOSIS
DN BR40:33992
TI PYRIMIDINE DERIVATIVES **PSYCHOTROPIC** PROPERTIES AND MOLECULAR MECHANISMS OF CENTRAL ACTION.

AU KARKISHCHENKO N N; MAKLYAKOV YU S; STRADOMSKII B V
CS DIV. PHARMACOL. CLIN. PHARMACOL., ROSTOV MED. INST., 344022 ROSTOV-NA-DONU, USSR.
SO Farmakol. Toksikol. (Moscow), (1990) 53 (4), 67-72.
CODEN: FATOAO. ISSN: 0014-8318.
FS BR; OLD
LA Russian
CC Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biophysics - Molecular Properties and Macromolecules *10506
Biophysics - Membrane Phenomena *10508
Endocrine System - Neuroendocrinology *17020
Nervous System - Physiology and Biochemistry *20504
Nervous System - Pathology *20506
Psychiatry - Psychopathology; Psychodynamics and Therapy *21002
Pharmacology - Drug Metabolism; Metabolic Stimulators *22003
Pharmacology - Neuropharmacology *22024
Pharmacology - Psychopharmacology *22026
BC Hominidae 86215
IT Miscellaneous Descriptors
REVIEW HUMAN URIDINE ANTIDEPRESSANT-DRUG TRANQUILIZER-DRUG BENZODIAZEPINE GAMMA AMINOBUTYRIC ACID IMIPRAMINE RECEPTOR PSYCHONEUROLOGICAL DISTURBANCE PHARMACODYNAMICS
RN 50-49-7 (IMIPRAMINE)
56-12-2 (GAMMA AMINOBUTYRIC ACID)
58-96-8 (URIDINE)
289-95-2D (PYRIMIDINE)
12794-10-4 (BENZODIAZEPINE)

L80 ANSWER 14 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1990:430316 BIOSIS
DN BA90:91117
TI **MEMORY** DEFICITS OF AGED MALE RATS CAN BE IMPROVED BY PYRIMIDINE NUCLEOSIDES AND N ACETYLGLUTAMINE.
AU DRAGO F; D'AGATA V; VALERIO C; SPADARO F; RAFFAELE R; NARDO L; GRASSI M; FRENI V
CS INST. PHARMACOL., FAC. MED., VIALE A. DORIA, 6 I-95125 CATANIA, ITALY.
SO CLIN NEUROPHARMACOL, (1990) 13 (4), 290-296.
CODEN: CLNEDB. ISSN: 0362-5664.
FS BA; OLD
LA English
AB The pyrimidine nucleosides uridine (URI) and cytidine (CYT), alone or associated with n-acetyl-glutamine (NAG), were injected acutely or subchronically to aged (26 months old) male rats of the Sprague-Dawley strain. Learning and memory abilities of the animals were studied with tests of avoidance behavior. The acquisition of active avoidance behavior was studied with the shuttle-box test. A step-through type of passive avoidance task was used to examine the retention of passive avoidance responses. The acquisition of the active avoidance behavior and the retention of the passive avoidance response were reduced in aged animals as compared with those of young animals. Neither the acute treatment of old rats with URI and CYT alone nor that associated with NAG exerted any effect on the behavioral tests. In contrast, the subchronic treatment with URI and CYT was followed by a facilitation of acquisition of active avoidance behavior in the shuttle box and of retention of passive avodiance responses in the dark box. A more potent effect on the acquisition of the shuttle-box behavior and on the retention of passive avoidance reaction was found in animals treated subchronically with the pyrimidine nucleosides associated with NAG. These effects may be related to the role of pyrimidines in the synthesis of ribonucleic acid, which is indispensable for learning and memory processes.
CC **Behavioral Biology - Animal Behavior *07003**
Behavioral Biology - Conditioning *07005
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Pathology, General and Miscellaneous - Therapy *12512
Psychiatry - General; Medical Psychology and Sociology *21001

Pharmacology - Clinical Pharmacology 22005
Pharmacology - Neuropharmacology *22024
Pharmacology - Psychopharmacology *22026
BC Muridae 86375
IT Miscellaneous Descriptors
URIDINE CYTIDINE CENTRAL STIMULANT-DRUG PASSIVE AVOIDANCE RESPONSE LEARNING ANIMAL MODEL
RN **58-96-8** (URIDINE)
65-46-3 (CYTIDINE)
289-95-2 (PYRIMIDINE)
2490-97-3Q, 35305-74-9Q (ACETYLGLUTAMINE)

L80 ANSWER 15 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1989:388595 BIOSIS
DN BA88:69185
TI EFFECTS OF **CHRONIC TREATMENT** WITH URIDINE ON STRIATAL DOPAMINE RELEASE AND DOPAMINE RELATED BEHAVIORS IN THE ABSENCE OR THE PRESENCE OF **CHRONIC TREATMENT** WITH HALOPERIDOL.
AU AGNATI L F; FUXE K; RUGGERI M; PICH E M; BENFENATI F; VOLTERRA V; UNGERSTEDT U; ZINI I
CS INST. HUM. PHYSIOL., UNIV. MODENA, VIA CAMPI 287, 41100 MODENA, ITALY.
SO NEUROCHEM INT, (1989) 15 (1), 107-114.
CODEN: NEUIDS. ISSN: 0197-0186.
FS BA; OLD
LA English
AB Uridine (15 mg/kg/day, i.p.), haloperidol (1 ng/kg/day, i.p.), uridine (15 mg/day, i.p.) plus haloperidol (1 mg/kg.day, i.p.) or saline have been chronically administered to Sprague-Dawley male rates. Following 1 week of wash-out, the effects of these treatments on basal striatal dopamine (DA) release as well as on the DA release induced by an acute haloperidol challenge (2 mg/kg, i.p.) were studied by means of intracerebral microdialysis. Behavioural tests such as haloperidol-induced catalepsy or apomorphine-induced stereotypies were also performed 4-7 days after drug withdrawal. The chronic treatment with uridine alone or associated with haloperidol markedly reduced DA release induced by an acute haloperidol challenge. The behavioural studies also indicated a change in DA-related behaviours in these conditions. The animals chronically treated with uridine showed significant increases in the stereotypy scores and in the catalepsy induced by an acute haloperidol challenge with respect to saline treated rats. The present results indicate that a chronic uridine treatment is able to potentiate the reduction of the striatal DA transmission induced by acute and chronic haloperidol treatment. This finding suggests that possibility to reduce the neuroleptic dose in the treatment of schizophrenia by combining neuroleptic and uridine treatments, thus making it possible to relieve some of the side effects of neuroleptic therapy.
CC **Behavioral Biology - Animal Behavior *07003**
Biochemical Studies - General 10060
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Pathology, General and Miscellaneous - Therapy 12512
Metabolism - Proteins, Peptides and Amino Acids *13012
Endocrine System - Neuroendocrinology *17020
Nervous System - Physiology and Biochemistry *20504
Nervous System - Pathology *20506
Psychiatry - Psychopathology; Psychodynamics and Therapy *21002
Pharmacology - Neuropharmacology *22024
Pharmacology - Psychopharmacology *22026
Toxicology - Pharmacological Toxicology *22504
Laboratory Animals - General 28002
BC Muridae 86375
IT Miscellaneous Descriptors
RAT MODEL ANTIPSYCHOTIC AGENT NEUROLEPTIC THERAPY SCHIZOPHRENIA
RN 51-61-6 (DOPAMINE)
52-86-8 (HALOPERIDOL)
58-96-8 (URIDINE)

L80 ANSWER 16 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1989:279680 BIOSIS
DN BR37:4677
TI TREATMENT OF PATIENTS PTS WITH CLASSICAL GALACTOSEMIA G WITH **ORAL URIDINE**.
AU KAUFMAN F R; NG W G; XU Y K; GUIDICI T; KALEITA T A; DONNELL G N
CS USC SCH. MED., CHILDRENS HOSP. LOS ANGELES, CALIF.
SO JOINT **MEETING** OF THE AMERICAN PEDIATRIC SOCIETY AND THE SOCIETY FOR PEDIATRIC RESEARCH, WASHINGTON, D.C., USA, MAY 1-4, 1989. PEDIATR RES. (1989) 25 (4 PART 2), 142A.
CODEN: PEREBL. ISSN: 0031-3998.
DT Conference
FS BR; OLD
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Behavioral Biology - Human Behavior *07004
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Biochemical Studies - Carbohydrates 10068
Pathology, General and Miscellaneous - Diagnostic 12504
Pathology, General and Miscellaneous - Therapy 12512
Metabolism - Carbohydrates *13004
Metabolism - Nucleic Acids, Purines and Pyrimidines *13014
Metabolism - Metabolic Disorders *13020
Nervous System - Physiology and Biochemistry *20504
Nervous System - Pathology *20506
Psychiatry - General; Medical Psychology and Sociology *21001
Pharmacology - Drug Metabolism; Metabolic Stimulators *22003
Pharmacology - Clinical Pharmacology *22005
BC Hominidae 86215
IT Miscellaneous Descriptors
ABSTRACT METABOLIC-DRUG PHARMACOKINETICS NEUROPSYCHOLOGY
RN **58-96-8** (URIDINE)

L80 ANSWER 17 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1988:356511 BIOSIS
DN BA86:51989
TI EFFECTS OF SHORT-TERM ADMINISTRATION OF CYTIDINE URIDINE AND L GLUTAMINE ALONE OR IN COMBINATION ON THE CEREBRAL ELECTRICAL ACTIVITY OF PATIENTS WITH **CHRONIC CEREBROVASCULAR** DISEASE.
AU MANNA V; MARTUCCI N
CS DEP. NEUROL. NEUROPHYSIOPATHOL., ITALIAN INST. NEUROTRAUMATOL., GROTTAFERRATA, ROME, ITALY
SO INT J CLIN PHARMACOL RES, (1988) 8 (3), 199-210.
CODEN: CPHRDE. ISSN: 0251-1649.
FS BA; OLD
LA English
AB Various data in the literature confirm that spectral analysis by an electroencephalograph (EEG) allows the evaluation and quantification of the modifications of cerebral electrical activity due to pathological events or to drugs acting on the central nervous system. Thirty patients with chronic cerebrovascular disease, selected according to the criteria established by the 1980 Paris Ad Hoc Committee were submitted to EEG analysis in basal conditions and after intravenous short-term administration of various doses of cytidine, uridine and levoglutamine given either alone or in combination. The combined administration of the three substances led to a general improvement of cerebral electrical parameters with a trend toward more physiological patterns.
CC Biochemical Studies - General 10060
Pathology, General and Miscellaneous - Therapy 12512
Cardiovascular System - Blood Vessel Pathology *14508
Nervous System - Pathology *20506
Pharmacology - Clinical Pharmacology *22005
Pharmacology - Cardiovascular System *22010
Pharmacology - Neuropharmacology *22024
BC Hominidae 86215

IT Miscellaneous Descriptors
CENTRAL NERVOUS SYSTEM
RN 56-85-9 (L GLUTAMINE)
58-96-8 (URIDINE)
65-46-3 (CYTIDINE)

L80 ANSWER 18 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1988:257899 BIOSIS
DN BR34:128929
TI URIDINE AS A POSSIBLE TREATMENT FOR AMYOTROPHIC LATERAL **SCLEROSIS** ALS HYPOTHESIS AND PHASE-I STUDY DEMONSTRATING SAFETY.
AU ENGEL W K
CS LOS ANGELES, CALIF.
SO 40TH ANNUAL **MEETING** OF THE AMERICAN ACADEMY OF NEUROLOGY, CINCINNATI, OHIO, USA, APRIL 17-23, 1988. NEUROLOGY. (1988) 38 (3 SUPPL 1), 326.
CODEN: NEURAI. ISSN: 0028-3878.
DT Conference
FS BR; OLD
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Genetics and Cytogenetics - Human *03508
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Pathology, General and Miscellaneous - Therapy 12512
Metabolism - Nucleic Acids, Purines and Pyrimidines *13014
Muscle - Pathology *17506
Nervous System - Pathology *20506
Pharmacology - Drug Metabolism; Metabolic Stimulators *22003
Pharmacology - Clinical Pharmacology *22005
Pharmacology - Neuropharmacology *22024
BC Hominidae 86215
IT Miscellaneous Descriptors
ABSTRACT HUMAN METABOLIC-DRUG DNA RNA SPINAL MUSCULAR ATROPHY
RN **58-96-8** (URIDINE)

L80 ANSWER 19 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1987:48021 BIOSIS
DN BA83:27367
TI EFFECTS OF LARGE DOSES OF PYRIMIDINE NUCLEOSIDES CYTIDINE AND URIDINE IN ELDERLY PATIENTS WITH **NEUROPSYCHOLOGICAL** DISTURBANCES CAUSED BY VASCULAR AND CEREBRAL METABOLIC INSUFFICIENCY.
AU MERLINI G; BREDA D; VALVERI G; ZILIOTTO G
CS VIA VENDRAMINI, 7-35100 PADOVA.
SO GAZZ MED ITAL ARCH SCI MED, (1986) 145 (6), 379-390.
CODEN: GMIMES.
FS BA; OLD
LA Italian
AB The effects of large doses of endovenous and intramuscular nucleosides (Cytidine and Uridine) were studied in a double blind test against a placebo conducted on a group of elderly patients hospitalized with substantial mental deterioration related to aging. Analysis of the results of the psychometric tests employed showed a statistically significant improvement among the treated patients in relation to memorisation process, attention, concentration capacity, temporospatial orientation and constructive praxis as well as diminishing depression, anxiety and related disturbances. The result was an improvement in individual performances, easier interpersonal communication and an improved quality of life. The drugs were always very well tolerated.
CC Biochemical Studies - General 10060
Cardiovascular System - Blood Vessel Pathology *14508
Nervous System - Pathology *20506
Psychiatry - Psychophysiology *21003
Pharmacology - Neuropharmacology *22024
Pharmacology - Psychopharmacology *22026
Gerontology *24500

BC Hominidae 86215
IT Miscellaneous Descriptors
AGING PHARMACODYNAMICS
RN **58-96-8** (URIDINE)
65-46-3 (CYTIDINE)
289-95-2 (PYRIMIDINE)

L80 ANSWER 20 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1986:154646 BIOSIS
DN BA81:65062
TI **SLEEP**-PROMOTING EFFECTS OF INTRAPERITONEALLY ADMINISTERED URIDINE IN UNRESTRAINED RATS.
AU HONDA K; OKANO Y; KOMODA Y; INOUE S
CS INST. MED. DENTAL ENG., TOKYO MED. DENTAL UNIV., KANDA-SURUGADAI 2-3-10, CHIYODA-KU, TOKYO 101.
SO NEUROSCI LETT, (1985) 62 (1), 137-142.
CODEN: NELED5. ISSN: 0304-3940.
FS BA; OLD
LA English
AB An intraperitoneal injection of 0.1 nmol uridine in rats resulted in a transient excess slow-wave sleep if administered shortly before onset of the dark period. The sleep latency was remarkably shortened. A small dose (0.01 nmol) and larger doses (1, 10, 100 nmol) caused no effect. Uridine at a dose of 0.1 nmol was entirely ineffective if injected shortly before onset of the light period, while it resulted in transient exess parodoxical sleep if injected at an early phase of the light period. It is concluded that uridine, if timely administered through a systemic route, may pass the blood-brain barrier to modulate sleep in rats.
CC **Behavioral Biology - Animal Behavior *07003**
Circadian Rhythms and Other Periodic Cycles *07200
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biophysics - Membrane Phenomena *10508
Blood, Blood-Forming Organs and Body Fluids - Blood and Lymph Studies *15002
Nervous System - Physiology and Biochemistry *20504
Psychiatry - Psychophysiology *21003
Pharmacology - Neuropharmacology *22024
Routes of Immunization, Infection and Therapy 22100
BC Muridae 86375
IT Miscellaneous Descriptors
BLOOD-BRAIN BARRIER LIGHT PERIOD DARK PERIOD
RN **58-96-8** (URIDINE)

L80 ANSWER 21 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1986:107694 BIOSIS
DN BA81:18110
TI A COMPARISON OF THE DOSE RESPONSE EFFECTS OF PYRIMIDINE RIBONUCLEOSIDES AND ADENOSINE ON **SLEEP** IN RATS.
AU RADULOVACKI M; VIRUS R M; RAPOZA D; CRANE R A
CS DEPARTMENT PHARMACOLOGY, COLLEGE MEDICINE, UNIVERSITY ILLINOIS CHICAGO, 835 W WOLCOTT AVENUE, CHICAGO, ILL. 60612, USA.
SO PSYCHOPHARMACOLOGY, (1985) 87 (2), 136-140.
CODEN: PSCHDL. ISSN: 0033-3158.
FS BA; OLD
LA English
AB The dose-response effects of intracerebroventricular (ICV) infusion of the pyrimidine ribonucleosides cytidine and uridine and the purine ribonucleoside adenosine on sleep and wakefulness (W) in rats were examined and compared. All three drugs were administered at doses of 1, 10, and 100 nmol in volumes of 5 .mu.l, with control animals receiving equivolumetric infusions of 0.9% saline. Treatment with 1 nmol cytidine significantly increased W and decreased both deep slow wave sleep (S2) and total sleep (TS) during both the 3-6 and 0-6 h recording periods. In addition, this dose of cytidine significantly increased light slow wave sleep (S1) during the first 3 h of recording. The 10 nmol dose of cytidine increased W and decreased TS during the 0-6 h recording. ICV

administration of uridine produced no significant changes in sleep and W at any dose during any of the recording periods examined. In contrast, adenosine exhibited significant hypnotic effects at all doses examined. All three doses of adenosine significantly reduced W and increased TS during both the 0-3 and 0-6 h recording periods. The 1 and 100 nmol doses of adenosine also significantly increased S2 during both the 0-3 and 0-6 h periods. In addition, the 100 nmol dose of adenosine significantly decreased W and increased both S2 and TS during the second 3 h of recording. Both the 1 and 100 nmol doses of adenosine also significantly reduced the latencies to the onset of rapid eye movement (REM) sleep. These data demonstrate the pyrimidine ribonucleosides do not produce hypnotic effects similar to those of adenosine and, in the case of cytidine, can actually suppress sleep. In addition, adenosine has been shown to significantly increase TS, primarily through an enhancement of S2, and decrease W at a dose as low as 1 nmol. The implications of these results with respect to the role of ribonucleosides, particularly adenosine, in the processes of sleep and W are discussed.

CC **Behavioral Biology - Animal Behavior 07003**
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Nervous System - Physiology and Biochemistry *20504
Psychiatry - Psychophysiology *21003
Pharmacology - Clinical Pharmacology 22005
Pharmacology - Neuropharmacology *22024
Pharmacology - Psychopharmacology *22026

BC Muridae 86375

IT Miscellaneous Descriptors
URIDINE CYTIDINE HYPNOTIC

RN 58-61-7 (ADENOSINE)
58-96-8 (URIDINE)
65-46-3 (CYTIDINE)
289-95-2 (PYRIMIDINE)

L80 ANSWER 22 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS

AN 1985:364533 BIOSIS

DN BA80:34525

TI **PROTECTIVE** EFFECT OF URIDINE ON D GALACTOSAMINE-INDUCED DEFICIENCY IN BRAIN URIDINE PHOSPHATES.

AU POPOV N; SCHMIDT S; MATTHIES H

CS INST. FUER PHARMAKOL. TOXIKOL. DER MED. AKAD. MAGDEBURG, 3090 MAGDEBURG, DDR.

SO BIOMED BIOCHIM ACTA, (**1984 (RECD 1985)**) 43 (12), 1399-1404. CODEN: BBIADT.

FS BA; OLD

LA English

AB In the rat, 2 h after intraventricular application of 10 .mu.mole D-galactosamine as well as 10 and 20 .mu.mol uridine, opposite effects on brain content of UDP-glucose and uracil nucleotides were observed. While D-galactosamine caused a strong decrease in content of uridine phosphates, the brain content of the latter substances was markedly increased after uridine application. Furthermore, 20 .mu.mol uridine applied 10 min prior to D-galactosamine administration prevented the D-galactosamine-induced drop in brain uridine phosphates. The results are discussed in the light of behavioral findings in which D-galactosamine-induced impairment of retention performance of an acquired behavior could be abolished by uridine pretreatment.

CC **Behavioral Biology - Animal Behavior *07003**
Biochemical Studies - General 10060
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Carbohydrates 10068
Enzymes - Physiological Studies *10808
Metabolism - Carbohydrates *13004
Metabolism - Proteins, Peptides and Amino Acids *13012
Metabolism - Nucleic Acids, Purines and Pyrimidines *13014
Nervous System - General; Methods 20501
Nervous System - Pathology *20506

Psychiatry - Psychopathology; Psychodynamics and Therapy *21002
Pharmacology - Neuropharmacology *22024
Pharmacology - Psychopharmacology *22026
Routes of Immunization, Infection and Therapy 22100
Toxicology - General; Methods and Experimental 22501
BC Muridae 86375
IT Miscellaneous Descriptors
RAT RETENTION UDP GLUCOSE URACIL
RN 50-99-7 (GLUCOSE)
58-96-8 (URIDINE)
58-98-0 (UDP)
66-22-8 (URACIL)
7535-00-4 (D GALACTOSAMINE)

L80 ANSWER 23 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1985:244403 BIOSIS
DN BA79:24399
TI LITTLE **SLEEP**-PROMOTING EFFECT OF 3 **SLEEP** SUBSTANCES DIURNALLY INFUSED IN UNRESTRAINED RATS.
AU INOUE S; HONDA K; KOMODA Y; UCHIZONO K; UENO R; HAYAISHI O
CS INSTITUTE FOR MEDICAL AND DENTAL ENGINEERING, TOKYO MEDICAL AND DENTAL UNIVERSITY, KANDA-SURUGADAI 2-3-10, CHIYODA-KU, TOKYO 101.
SO NEUROSCI LETT, (1984) 48 (1-2), 207-212.
CODEN: NELED5. ISSN: 0304-3940.
FS BA; OLD
LA English
AB Delta-sleep-inducing peptide (2.5 nmol), prostaglandin D2 (0.36 nmol) and uridine (10 pmol) were infused i.v. for 10 h at daytime in otherwise saline-infused freely moving male rats. In contrast to a nocturnal infusion which may result in marked sleep-promoting effects, such a diurnal infusion brought about almost no change in sleep parameters. The requirement of sleep in rats might be fully achieved at the environmental light period to cancel the effect of the exogenously administered sleep substances. An endogenous sleep substance should be characterized by a property not to cause excessive sleep at the time when sleep is physiologically saturated.
CC **Behavioral Biology - Animal Behavior *07003**
Circadian Rhythms and Other Periodic Cycles *07200
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Lipids 10066
External Effects - Light and Darkness 10604
Endocrine System - General *17002
Nervous System - Physiology and Biochemistry *20504
Psychiatry - Psychophysiology *21003
Pharmacology - Neuropharmacology *22024
Pharmacology - Psychopharmacology *22026
Routes of Immunization, Infection and Therapy 22100
BC Muridae 86375
IT Miscellaneous Descriptors
DELTA-SLEEP-INDUCING PEPTIDE PROSTAGLANDIN D-2 URIDINE NOCTURNAL EFFECT
RN **58-96-8** (URIDINE)
41598-07-6 (PROSTAGLANDIN D-2)
69431-45-4 (DELTA-SLEEP-INDUCING PEPTIDE)

L80 ANSWER 24 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1980:150296 BIOSIS
DN BA69:25292
TI INFLUENCE OF SOME BIOLOGICAL PYRIMIDINES ON THE SUCCINATE CYCLE DURING AND AFTER CEREBRAL **ISCHEMIA** IN RATS.
AU BENZI G; ARRIGONI E; MARZATICO F; VILLA R F
CS INST. PHARMACOL., DEP. SCI., UNIV. PAVIA, PAVIA, ITALY.
SO BIOCHEM PHARMACOL, (1979) 28 (17), 2545-2550.
CODEN: BCPCA6. ISSN: 0006-2952.
FS BA; OLD
LA English

AB Some cortical metabolites (glycogen, glucose, G-6-P, pyruvate, lactate, .alpha.-ketoglutarate, succinate, fumarate, malate, citrate, glutamate, glutamine, alanine, NH4+) were studied in rat brain after 5 min of complete compression ischemia, and after 15 min of recirculation following 5 min of ischemia. The 2 conditions (ischemia and post ischemic restitution) were induced in control animals and in rats pretreated 1 h before by an i.p. injection of 120 mg/kg of some biological pyrimidines (uridine, cytidine and uridine disphosphate glucose [UDPG]). At the cerebral level total complete ischemia induced the: drop of substrates and of glycolytic intermediates, consistent with the increase of lactate and redox state; increase of succinate and alanine, decrease of malate and fumarate and depletion of .alpha.-ketoglutarate. Some of the events may be regarded as the expression of the activation of the succinate cycle which contributed by .apprx. 10% to the release of anaerobic energy during cerebral ischemia. Pretreatment with the tested pyrimidines did not modify this cerebral biochemical pattern. During post-ischemic recovery, cerebral parameters tended to normalize, except for a further increase in alanine production (as an expression of the activation of the alanine aminotransferase reaction) with conversion of pyruvate into .alpha.-ketoglutarate available for the ammonia-detoxicating processes (amination to glutamate and amidation to glutamine). During post-ischemic recovery, pretreatment with cytidine was poorly active. Pretreatment with uridine decreased glucose, G-6-P and pyruvate cerebral concentrations, while succinate and alanine were increased. This latter effect was present in the case of pretreatment with UDPG. UDPG increased the cerebral concentration of glycogen and decreased those of fumarate and malate. The different biochemical actions of uracyl derivatives are discussed with regard to their biological effects.

CC Biochemical Studies - General 10060
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Carbohydrates 10068
Biophysics - General Biophysical Techniques 10504
Biophysics - Molecular Properties and Macromolecules *10506
Biophysics - Bioenergetics: Electron Transport and Oxidative Phosphorylation 10510
Enzymes - Chemical and Physical 10806
Enzymes - Physiological Studies *10808
Anatomy and Histology, General and Comparative - Experimental Anatomy 11104
Chordate Body Regions - Abdomen 11314
Movement 12100
Pathology, General and Miscellaneous - Therapy 12512
Metabolism - General Metabolism; Metabolic Pathways *13002
Metabolism - Energy and Respiratory Metabolism *13003
Metabolism - Carbohydrates *13004
Metabolism - Proteins, Peptides and Amino Acids *13012
Metabolism - Nucleic Acids, Purines and Pyrimidines *13014
Cardiovascular System - General; Methods 14501
Cardiovascular System - Physiology and Biochemistry 14504
Cardiovascular System - Blood Vessel Pathology *14508
Blood, Blood-Forming Organs and Body Fluids - Blood and Lymph Studies 15002
Nervous System - General; Methods 20501
Nervous System - Pathology *20506
Pharmacology - Drug Metabolism; Metabolic Stimulators *22003
Pharmacology - Cardiovascular System *22010
Pharmacology - Neuropharmacology *22024
Routes of Immunization, Infection and Therapy 22100
Toxicology - General; Methods and Experimental *22501

BC Muridae 86375

IT Miscellaneous Descriptors
CORTEX URIDINE CYTIDINE URIDINE DI PHOSPHATE GLUCOSE
CARDIOVASCULAR-DRUG GLYCOGEN GLUCOSE GLUCOSE 6 PHOSPHATE PYRUVATE
LACTATE ALPHA KETO GLUTARATE SUCCINATE FUMARATE MALATE GLUTAMINE
ALANINE AMMONIUM ION ALANINE AMINO TRANSFERASE REDOX STATE AMMONIA DE

TOXICATION PHARMACO KINETICS
RN 50-99-7 (GLUCOSE)
56-14-4 (SUCCINATE)
56-73-5 (GLUCOSE 6 PHOSPHATE)
57-60-3 (PYRUVATE)
58-96-8 (URIDINE)
65-46-3 (CYTIDINE)
113-21-3 (LACTATE)
133-89-1 (URIDINE DI PHOSPHATE GLUCOSE)
142-42-7 (FUMARATE)
149-61-1 (MALATE)
289-95-2D (PYRIMIDINES)
328-50-7 (ALPHA KETO GLUTARATE)
9000-86-6 (ALANINE AMINO TRANSFERASE)
9005-79-2 (GLYCOGEN)
14798-03-9 (AMMONIUM ION)
56-41-7Q, 6898-94-8Q (ALANINE)
56-85-9Q, 6899-04-3Q (GLUTAMINE)

L80 ANSWER 25 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1975:116850 BIOSIS
DN BA59:16850
TI RNA TREATMENT OF **DEMENTIA** A DOUBLE-BLIND STUDY.
AU MUNCH-PETERSEN S; PAKKENBERG H; KORNERUP H; ORTMANN J; IPSEN E; JACOBSEN P; SIMMELSGARD H
SO ACTA NEUROL SCAND, (1974) 50 (5), 553-572.
CODEN: ANRSAS. ISSN: 0001-6314.
FS BA; OLD
LA Unavailable
CC Methods, Materials and Apparatus, General - Photography 01012
Cytology and Cytochemistry - Animal 02506
Radiation - Radiation and Isotope Techniques 06504
Behavioral Biology - Animal Behavior 07003
Behavioral Biology - Human Behavior 07004
Biochemistry - Gases 10012
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Sterols and Steroids 10067
Biophysics - General Biophysical Techniques 10504
Anatomy and Histology, General and Comparative - Radiologic Anatomy 11106
Pathology, General and Miscellaneous - Therapy *12512
Metabolism - Sterols and Steroids *13008
Metabolism - Proteins, Peptides and Amino Acids *13012
Metabolism - Porphyrins and Bile Pigments *13013
Blood, Blood-Forming Organs and Body Fluids - Blood and Lymph Studies *15002
Dental and Oral Biology - General; Methods 19001
Nervous System - General; Methods 20501
Nervous System - Physiology and Biochemistry *20504
Nervous System - Pathology *20506
Psychiatry - General; Medical Psychology and Sociology 21001
Psychiatry - Psychopathology; Psychodynamics and Therapy *21002
Pharmacology - Drug Metabolism; Metabolic Stimulators *22003
Pharmacology - Clinical Pharmacology 22005
Routes of Immunization, Infection and Therapy 22100
Plant Physiology, Biochemistry and Biophysics - Chemical Constituents 51522
BC Fungi - Unspecified 15000
Hominidae 86215
Muridae 86375
IT Miscellaneous Descriptors
MOUSE HUMAN URIDINE UPTAKE
RN **58-96-8** (URIDINE)
58-96-8 (URIDINE)

L80 ANSWER 26 OF 26 BIOSIS COPYRIGHT 2000 BIOSIS

AN 1971:174828 BIOSIS
DN BA52:84828
TI THE EFFECTS OF DRUGS ON **LEARNING** IN A SIMPLE PREPARATION.
AU KERKUT G A; OLIVER G W O; RICK J T; WALKER R J
SO COMP GEN PHARMACOL, (1970) 1 (4), 437-483.
CODEN: CPGPAY. ISSN: 0010-4035.
FS BA; OLD
LA Unavailable
CC Radiation - Radiation and Isotope Techniques 06504
Behavioral Biology - Conditioning *07005
Biochemical Studies - General 10060
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biophysics - General Biophysical Techniques 10504
Enzymes - Physiological Studies *10808
Movement 12100
Metabolism - Proteins, Peptides and Amino Acids *13012
Metabolism - Nucleic Acids, Purines and Pyrimidines *13014
Nervous System - Physiology and Biochemistry *20504
Pharmacology - Drug Metabolism; Metabolic Stimulators *22003
Pharmacology - Neuropharmacology *22024
Pharmacology - Psychopharmacology 22026
Invertebrata, Comparative and Experimental Morphology, Physiology and Pathology - Insecta - Physiology *64076
Invertebrate Body Regions and Structures - Appendages 64212
BC Orthoptera 75340
IT Miscellaneous Descriptors
COCKROACH METAB-DRUGS RNA PROTEIN SYNTHESIS METATHORACIC GANGLION URIDINE LEUCINE INCORPORATION RADIOACTIVE LABEL CHOLIN ESTERASE
RN **58-96-8** (URIDINE)
9001-08-5 (CHOLIN ESTERASE)
61-90-5Q, 7005-03-0Q (LEUCINE)

=> fil wpids

FILE 'WPIDS' ENTERED AT 09:57:12 ON 08 AUG 2000
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=> d his l81-

(FILE 'BIOSIS' ENTERED AT 08:52:37 ON 08 AUG 2000)

FILE 'WPIDS' ENTERED AT 08:53:07 ON 08 AUG 2000
L81 931 S URIDINE
E URIDINE/DCN
E E3+ALL/DCN
L82 129 S E2 OR 0177/DRN

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L83         976 S L81,L82
L84          55 SEA L83 AND (P440 OR P444 OR P445 OR P446 OR P447 OR P448 OR
                P450 OR P451 OR P517 OR P528 OR P520)/M0,M1,M2,M3,M4,M5,M6
L85          15 S L83 AND (B14-J? OR C14-J?)/MC
L86          54 S L83 AND (B12-C? OR C12-C?)/MC
L87          25 S L83 AND (B12-D? OR C12-D?)/MC
L88          22 S L83 AND (B12-E? OR C12-E?)/MC
L89           0 S L83 AND (B12-F07? OR C12-F07?)/MC
L90          23 S L83 AND (B14-N16 OR C14-N16 OR B12-C10 OR C12-C10)/MC
L91         110 S L84-L90
L92          63 S L91 AND URID?/TI
L93          47 S L91 NOT L92
L94          11 S L92 AND (PSYCHO? OR STRESS OR PARKINSON? OR MEMORY OR DEGENER
                E WATKINS C/AU
L95          66 S E3-E14
                E WURTMAN R/AU
L96          53 S E3,E4
L97           1 S L95,L96 AND L83
L98          11 S L94,L97

     FILE 'WPIDS' ENTERED AT 09:57:12 ON 08 AUG 2000

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L98  ANSWER 1 OF 11  WPIDS COPYRIGHT 2000   DERWENT INFORMATION LTD
AN   2000-195185 [17]   WPIDS
DNC  C2000-060486
TI   Treatment of cytidine-dependent neurological disorders e.g. memory
     disorders, cognitive dysfunction, emotional disorders, ataxia, tardive
     dyskinesia and cerebral thrombosis by administration of uridine
     or a uridine source.
DC   B03
IN   WATKINS, C; WURTMAN, R J
PA   (MASI) MASSACHUSETTS INST TECHNOLOGY
CYC  20
PI   WO 2000006174 A1 20000210 (200017)* EN   22p    A61K031-55
        RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE
         W: CA JP
ADT  WO 2000006174 A1 WO 1999-US17235 19990730
PRAI US 1998-95002    19980731
IC   ICM  A61K031-55
     ICS  A61K031-235; A61K031-515; A61K031-685; A61K031-70
AB   WO 200006174 A UPAB: 20000405
     NOVELTY - A new method of treating neurological disorders comprises
     administration of uridine or a uridine source.
          DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for a method
     for treating neurological disorders comprising administration of at least
     uridine or a uridine source and other compounds.
          ACTIVITY - Neuroprotective; nootropic; anticonvulsant; tranquilizer;
     antidepressant; thrombolytic; vasotropic. Gerbils were given orally
     uridine (250 mg/kg orally) and 60 minutes later plasma and brain
     levels of cytidine and uridine were measured by HPLC method.
     Plasma levels of uridine were raised by 100 % and cytidine by
     13.9% while brain levels of uridine were raised by 100 % and
     cytidine levels by 39%Results indicated that uridine, when
     transported to the brain, was readily converted to cytidine (conversion
     more efficient in the brain than in the plasma). Similar experiments were
     conducted in humans showed similar results (by measuring the CSF levels).
          MECHANISM OF ACTION - Increases systemic and brain cytidine levels.
          USE - The method is useful for treating neurological disorders,
     especially memory disorders (including memory decline associated with
     brain aging, e.g. Pick's disease, Lewy Body disease, Huntington's disease
     and AIDS dementia), cognitive dysfunction (including lack of attention,
     alertness, concentration, focus or dyslexia), emotional disorders
     (including mania, depression, stress, panic, anxiety, dysthymia,
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psychosis, seasonal affective disorder and bipolar disorder), ataxia, Friedreich's ataxia, tardive dyskinesia, cerebral thrombosis, ischemia, cerebrovascular diseases resulting from hypoxia, behavioral and neurological syndromes (including after brain trauma, spinal cord injury and anoxia) and peripheral nervous system disorders (including myasthenia gravis, post polio syndrome and muscular dystrophy).

ADVANTAGE - Administration of **uridine** elevates levels of cytidine in the brain and is transported across the blood-brain barrier much more efficiently.

Dwg.0/4

FS CPI

FA AB; DCN

MC CPI: B04-B01B; B04-B03A; B05-B01P; B07-D12; B07-D13; B10-A22; B10-B02H; B10-C04E; B14-F01E; B14-F02C; B14-F02D; B14-F04; **B14-J01**; **B14-J01A1**; **B14-J01A4**; **B14-J02**; **B14-J07**; **B14-N16**

TECH UPTX: 20000405

TECHNOLOGY FOCUS - PHARMACEUTICALS - Preferred Drugs: The other compound is preferably choline (especially choline chloride, choline bitartrate and choline stearate), a choline precursor (especially sphingomyelin, cytidine-diphospho-choline, citicoline or CDP-choline, an acylglycerophosphocholine, lecithin, lysolecithin, glycerophosphatidylcholine or fatty acids), **uridine** phosphorylase inhibitors (e.g. benzyl barbiturate), **uridine** secretion inhibitors (dilazep or hexobendine), **uridine** renal transport competitors (L-**uridine**, L-2',3'-dideoxyuridine and D-2',3'-dideoxyuridine).

L98 ANSWER 2 OF 11 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD

AN 1998-032325 [03] WPIDS

DNC C1998-010921

TI Composition comprising **uridine** and neurotrophins - useful in treatment of disturbances of nervous system due to **degeneration** of neuronal or glial cells.

DC B03 B04

IN MATERAZZI, M; PIAZZA, C; POLITI, V

PA (POLI-N) POLIFARMA SPA

CYC 77

PI WO 9745127 A1 19971204 (199803)* EN 27p A61K031-70

RW: AT BE CH DE DK EA ES FI FR GB GH GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG

W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT UA UG UZ VN YU

AU 9730468 A 19980105 (199821) A61K031-70

EP 914131 A1 19990512 (199923) EN A61K031-70

R: AT CH DE DK ES FI FR GB IT LI NL PT SE

US 5962459 A 19991005 (199948)# A61K031-55

BR 9709379 A 20000111 (200020) A61K031-70

JP 2000503668 W 20000328 (200026) 22p A61K031-70

HU 2000000124 A2 20000528 (200035) A61K031-70

ADT WO 9745127 A1 WO 1997-IT117 19970523; AU 9730468 A AU 1997-30468 19970523; EP 914131 A1 EP 1997-925266 19970523, WO 1997-IT117 19970523; US 5962459 A Provisional US 1996-18543 19960529, US 1997-862306 19970523; BR 9709379 A BR 1997-9379 19970523, WO 1997-IT117 19970523; JP 2000503668 W JP 1997-541970 19970523, WO 1997-IT117 19970523; HU 2000000124 A2 WO 1997-IT117 19970523, HU 2000-124 19970523

FDT AU 9730468 A Based on WO 9745127; EP 914131 A1 Based on WO 9745127; BR 9709379 A Based on WO 9745127; JP 2000503668 W Based on WO 9745127; HU 2000000124 A2 Based on WO 9745127

PRAI IT 1996-RM364 19960528; US 1997-862306 19970523

IC ICM A61K031-55; A61K031-70

ICS A61P025-00; A61P025-16; A61P025-28; A61P031-00; A61P035-00

AB WO 9745127 A UPAB: 19980119

Composition (I), for the treatment of disturbances of the nervous system due to selective degeneration of neuronal or glial cells, comprises

uridine and neurotrophins. The ratio of **uridine** :neurotrophins is 1:10 to 1:100. Also claimed is the use of **uridine** as a substance for mimicking neurotrophins in a treatment as above.

USE - (I) are used in the treatment of disturbances of the nervous system, and also for promoting differentiation and functioning and maturation of cells. It may be used in treatment of nervous system disturbances due to degeneration of neuronal/glial cells, such as peripheral neuropathies of iatrogenic origin (e.g. as a consequence of administration of anti-viral drugs, especially those used in the treatment of AIDS, or anti-tumour drugs), Alzheimer's disease, Parkinson's disease, stroke, lateral amyotrophic sclerosis (all claimed).

Dwg.0/0

FS CPI
FA AB; GI; DCN
MC CPI: B04-B03A; B04-H06; **B14-J01**

L98 ANSWER 3 OF 11 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD
AN 1995-317412 [41] WPIDS
DNC C1995-140870
TI Drug compsn. for enhancing resistance to **psychological stress** - comprises nucleic acid mixt. of inosine, cytidine, **uridine**, guanosine-5'-phosphate or its salt, and thymidine..
DC B02 B03
PA (SAKA) OTSUKA SEIYAKU KOGYO KK
CYC 1
PI JP 07215879 A 19950815 (199541)* 4p A61K031-70
ADT JP 07215879 A JP 1994-10774 19940202
PRAI JP 1994-10774 19940202
IC ICM A61K031-70
AB JP 07215879 A UPAB: 19951019

Drug compsn. comprises a mixt. of inosine, cytidine, **uridine**, guanosine-5'-phosphate or its salt, and thymidine as active ingredients.

The molar ratio of the five components is pref. inosine/ cytidine/ **uridine**/ guanosine-5'-phosphate or its salt/ thymidine = 4:4:3:4:1. Na or K salt, pref. disodium salt.

For intravenous admin., an injection contg. the mixt. at concn. of 2 - 8 %(w/v) is suitable. For oral admin. the daily dose is pref. 1.5 - 2.5 g/patient.

USE/ADVANTAGE - The compsn. is useful for the prevention and/or treatment of various syndromes induced by excessive psychological stress, e.g. digestive ulcer. It has less serious side effects (i.e. a hepatic coma) than tyrosine, a drug known for its anti-stress activity.

A gp. of male S-D rats was fed with 20% casein contg. a 4:4:3:4:1 (in mole) mixt. of inosine, cytidine, **uridine**, guanosine-5'-phosphate disodium salt, and thymidine at 0.5 %(w/w) for 14 days. After starvation for 24 hours they were deprived of body movement by restriction and sunk in water except their heads for three hours to induce stress ulcer. Immediately afterwards their stomachs were extracted and the ratio of the ulcer area to the total area of the gastric mucous membrane was measured as a stress indicator. The rats fed with a diet contg. the drug showed the ratio of 1.73 %, significantly lower than the ratio (7.08 %) for rats fed only 20% casein.

Dwg.0/0

FS CPI
FA AB; DCN
MC CPI: B04-B03A; B04-B03B; B14-E08

L98 ANSWER 4 OF 11 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD
AN 1994-089979 [11] WPIDS
DNC C1994-041613
TI Use of **uridine** as **antidepressant** - shows its reduced toxicity and elimination of several harmful side effects.
DC B03
IN KARKISHCHENKO, N N; MAKLYAKOV, YU S; STRADOMSKII, B V
PA (ROME) ROST MED INST

CYC 1
PI RU 2003332 C1 19931130 (199411)* 4p A61K031-505
ADT RU 2003332 C1 SU 1989-4702930 19890609
PRAI SU 1989-4702930 19890609
IC ICM A61K031-505
AB RU 2003332 C UPAB: 19940428
Novel use of **uridine** (I) as an antidepressant. (I) is an N-glycoside of uracil in which the first C atom of ribose is joined, through the glycoside bond, to N-1 of uracil. It is an endogenic cpd. which appears in nucleic acids and in some ferments.
USE/ADVANTAGE - In treatment of depression. Increased activity, fewer side effects, reduced toxicity.
FS CPI
FA AB; DCN
MC CPI: B04-B03A; **B14-J01A1**

L98 ANSWER 5 OF 11 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD
AN 1993-128387 [16] WPIDS
DNC C1993-056960
TI New **uridine** deriv. **CNS** inhibitors - are hypnotics, **anxiolytics** and anti-epileptics etc. obtd. by reacting N-unsubstituted **uridine**(s) with phenacyl halide derivs. in presence of base.
DC B03
PA (NISR) NISSUI PHARM CO LTD
CYC 1
PI JP 05059087 A 19930309 (199316)* 7p C07H019-06
JP 3032053 B2 20000410 (200023) 8p C07D405-04
ADT JP 05059087 A JP 1991-227230 19910906; JP 3032053 B2 JP 1991-227230 19910906
FDT JP 3032053 B2 Previous Publ. JP 05059087
PRAI JP 1991-227230 19910906
IC ICM C07D405-04; C07H019-06
ICS A61K031-506; A61K031-70; A61K031-7072; A61P025-00; A61P025-20; A61P025-22
AB JP 05059087 A UPAB: 19930924
Uridine derivs. of formula (1) are new, where R' = H, halogen atom or lower alkyl, R2 and R3 = H, OH or lower alkoxy, R4 = H or lower alkyl.
(1) may be prepd. by reacting N-unsubstd. **uridine** derivs. of formula (2) (e.g. **uridine**, deoxyuridine, 2',3',5'- tri-O-methyluridine, 2',3',5'-tri-O-ethyluridine, 2',3',5'-tri-O -propyluridine, 3',5'-di-O-methyldeoxyuridine, 3',5'-di-O -ethyldeoxyuridine, 3',5'-di-O-propyldeoxyuridine with a phenacyl halide of formula (3) (where X = halogen atom) (e.g. phenacyl bromide, p-bromophenacyl bromide, p-methylphenacyl bromide) in presence of a base, e.g. K2CO3, Na2CO3 in an inert solvent, e.g. DMF, acetone at a temp. of 5-150 deg.C for 1 to 50 hrs.
USE/ADVANTAGE - (1) exhibit potent hypnotic action and action prolonging pentobarbital sleep with low toxicity and are useful as central inhibitors e.g. psychopharmaceuticals, anti-anxiety drugs, anti-epileptics, muscular relaxants. (1) may be administered orally as powder, tablets, capsules or granules at a dose of 5-20 mg/60 kg. Also applicable parenterally as injection (i.v. into ventriculus lateralis).
In an example, to a soln. of 4.8841 g. (20 mmol.) **uridine** and 4.7 g. (34 mmol.) K2CO3 in 16 ml. DMF and 16 ml. acetone was added 4.65 g. (30 mmol.) phenacyl chloride and the mixt. refluxed at 90 deg.C for 5 hrs. and then evapd. in vacuo. The residue was chromatographed on a silica gel column (200 g.) and eluted with CHCl3/EtOAc/MeOH (5:4:1) to give light yellow crystals, which were recrystallised from EtOH/hexane (1:2) to give 3.7962 g. (52.4% yield) of N3-phenacryluridine as light yellow needles. IR (KBr) Fig.1.
0/0
FS CPI
FA AB; GI; DCN
MC CPI: B04-B03A; **B12-C07**; **B12-C10**; **B12-D04**;

B12-E02

L98 ANSWER 6 OF 11 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD
AN 1990-001674 [01] WPIDS
DNC C1990-000740
TI Using **uridine** to control function of dopaminergic system - esp. for treating **schizophrenia** and **Parkinson**'s disease.
DC B03
IN DELUCA, G F; DISTAZIO, G; MATERAZZI, M; POLITI, V; DE, LUCA G; DI, STAZIO G
PA (POLI-N) POLIFARMA SPA; (PLIF-N) PLIFARMA SPA
CYC 15
PI EP 348360 A 19891227 (199001)* EN 5p
R: AT BE CH DE ES FR GB LI NL SE
JP 02045425 A 19900215 (199013)
US 4960759 A 19901002 (199042)
IT 1219667 B 19900524 (199213)
EP 348360 B1 19940119 (199403) EN 7p A61K031-70
R: AT BE CH DE ES FR GB LI NL SE
DE 68912419 E 19940303 (199410) A61K031-70
CA 1327003 C 19940215 (199412) A61K031-70
JP 06023109 B2 19940330 (199416) 5p A61K031-70
ADT EP 348360 A EP 1989-830264 19890614; JP 02045425 A JP 1989-150354 19890613; US 4960759 A US 1989-367615 19890619; IT 1219667 B IT 1988-48118 19880621; EP 348360 B1 EP 1989-830264 19890614; DE 68912419 E DE 1989-612419 19890614, EP 1989-830264 19890614; CA 1327003 C CA 1989-600276 19890524; JP 06023109 B2 JP 1989-150354 19890613
FDT DE 68912419 E Based on EP 348360; JP 06023109 B2 Based on JP 02045425
PRAI IT 1988-48118 19880621
REP 3.Jnl.Ref; A3...9143; EP 178267; EP 216133; No-SR.Pub; WO 8903837
IC A61K031-70; C07H019-06
ICM A61K031-70
ICS A61K031-445; C07H019-06
ICA C07H019-067
AB EP 348360 A UPAB: 19930928
Uridine is used for the manufacture of a medicament for controlling the function of the dopaminergic system, and thus for treating psychic disorders of schizophrenic and Parkinson's disease type. Tests illustrate that an administration of **uridine** has a selective effect on the protection of cholecystokinin in cerebral ageing; and that it blocks the side-effects of neuroleptic drugs, esp. haloperidol.
0/0
FS CPI
FA AB; DCN
MC CPI: B04-B03A; **B12-C04**; **B12-C10**; B12-G04
ABEQ US 4960759 A UPAB: 19930928
New treatment of Parkinson's disease and disorders due to altered functioning of cerebral dopaminergic system or altered modulation of dopamine release in brain tissue, e.g. schizophrenia, esp. those due to low level of cholecystokinin (CCK), comprises admin. of **uridine** which increases CCK level.
USE - CCK is a natural hormonal peptide which modulates dopaminergic receptor, but fails to enter CNS by systemic admin., but **uridine** does enter and admin. increases CCK levels which fall on ageing, and improves dopaminergic functions, while blocking side effects of neuroleptics.
ABEQ EP 348360 B UPAB: 19940303
Use of **uridine** for the mfr. of a medicament for the treatment of the Parkinson's disease.
Dwg.0/0

L98 ANSWER 7 OF 11 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD
AN 1989-150746 [20] WPIDS
CR 1989-150747 [20]; 1989-339766 [46]; 1993-045427 [05]; 1993-311619 [39]; 1994-217794 [26]; 1995-006689 [01]; 1996-019901 [02]; 1996-087513 [09]; 1997-065133 [06]; 1998-239258 [21]; 1998-376859 [32]

DNC C1989-066755
TI New **uridine**- and cytidine acyl derivs - used in treatment e.g. of hepatopathy, diabetes, heart disease, **cerebrovascular** disorders and **parkinson**'s disease.
DC B03
IN BAMAT, M K; VON BORSTEL, R; VON BORSTEL, R W; VONBORSTEL, R W
PA (PRON-N) PRO-NEURON INC
CYC 23
PI WO 8903837 A 19890505 (198920)* EN 69p
RW: AT BE CH DE FR GB IT LU NL SE
W: AU BR DK FI JP KR NO SU US
AU 8927899 A 19890523 (198939)
EP 339075 A 19891102 (198944) EN
R: AT BE CH DE FR GB IT LI LU NL SE
JP 02500372 W 19900208 (199012)
ZA 8900232 A 19900627 (199030)
EP 339075 B1 19930818 (199333) EN 39p C07H019-067
R: AT BE CH DE FR GB IT LI LU NL SE
DE 3883374 G 19930923 (199339) C07H019-067
CA 1321994 C 19930907 (199342) C07H019-067
EP 339075 A4 19900530 (199511)
JP 07228535 A 19950829 (199543) 22p A61K031-70
IL 88208 A 19961016 (199648) A61K031-70
US 5583117 A 19961210 (199704) 21p A61K031-70
JP 10001436 A 19980106 (199811) 25p A61K031-70
JP 2894610 B2 19990524 (199926) 31p C07H019-067
ADT WO 8903837 A WO 1988-US3823 19881027; EP 339075 A EP 1988-909932 19881027; JP 02500372 W JP 1988-509176 19881027; ZA 8900232 A ZA 1988-232 19881027; EP 339075 B1 EP 1988-909932 19881027, WO 1988-US3823 19881027; DE 3883374 G DE 1988-3883374 19881027, EP 1988-909932 19881027, WO 1988-US3823 19881027; CA 1321994 C CA 1988-581429 19881027; EP 339075 A4 EP 1988-909932 ; JP 07228535 A Div ex JP 1988-509176 19881027, JP 1994-303877 19881027; IL 88208 A IL 1988-88208 19881028; US 5583117 A Cont of US 1987-115929 19871028, Div ex US 1991-737913 19910729, US 1993-140475 19931025; JP 10001436 A Div ex JP 1988-509176 19881027, JP 1997-36734 19881027; JP 2894610 B2 JP 1988-509176 19881027, WO 1988-US3823 19881027
FDT EP 339075 B1 Based on WO 8903837; DE 3883374 G Based on EP 339075, Based on WO 8903837; JP 2894610 B2 Previous Publ. JP 02500372, Based on WO 8903837
PRAI US 1987-115929 19871028; US 1991-737913 19910729; US 1993-140475 19931025
REP 1.Jnl.Ref; EP 178267; EP 222192; EP 56265; JP 52023085; JP 55024150; JP 57023085; JP 58049315; US 3585188; US 3894000; US 3975367; US 3991045; No-Citns.; JP 57091995
IC A61K031-70; C07D000-00; C07H019-06
ICM A61K031-70
ICS C07D000-00; C07H019-06
ICA C07H019-067
AB WO 8903837 A UPAB: 20000313
Acyl derivs. of **uridine** of formulae (I) and (II) and pharmaceutically acceptable salts are new In (I): R1-3 = H or acyl radical of a metabolite; and R4 = acyl radical of a metabolite. In (II): R1-3 = H or acyl of: (a) an unbranched 5-22C fatty acid; (b) an amino acid from Gly, Ala, Val, Leu, Ile, Tyr, Pro, L-hydroxyproline, Ser, Thr, Cys, Cys-Cys, Asp, Glu, Arg, Lys, His L-carnitine and Orn; (c) a 3-22C dicarboxylic acid; or (d) one or more of glycolic, pyruvic, lactic, enolpyruvic, lipoic, pantothenic, acetoacetic, p-aminobenzoic, beta-hydroxybutyric and orotic acids and creatine; provided that at least one of R1-3 is not H, and further that if any of R1-3 = H and if the remaining substits. are n-fatty acyl, then the n-fatty acyl contains 8-22.

Delivery of exogenous cytidine to animal tissue comprises admin. of an acyl deriv. of cytidine of formula (III), or salt: where R1-4 = H or acyl deriv. of a metabolite, provided at least one is not H.

USE/ADVANTAGE - Used for treating physiological or pathological conditions by supporting tissue metabolic functions, partic. in treatment of cardiac insufficiency and myocardial infarction, liver disease or

damage, muscle performance, lung disorders, diabetes, CNS disorders such as cerebrovascular disorders, Parkinson's disease and senile dementias, and to improve immune responses. The cpds. improve the bioavailability of **uridine** and cytidine by enhancing transport across the gastrointestinal tract and other biological membranes, and prevent their premature degradation. The acylated derivs. are effective when admin. p.o. Doses are e.g. 0.5-3.0 g (as nucleoside)/day. The **uridine** and cytidine derivs. may be coadmin.
Dwg.0/11

FS CPI
FA AB; DCN
MC CPI: B04-B03A; **B12-C04**; **B12-D02A**; B12-F01B; B12-G02; B12-G04A; B12-H05; B12-K06
ABEQ EP 339075 B UPAB: 19931119
An acyl derivative of **uridine** having the formula (II) wherein R1, R2, and R3 are the same or different and each is hydrogen or an acyl radical of (a) an unbranched fatty acid with 5 to 22 carbon atoms, (b) an amino acid selected from the group consisting of glycine, L-forms of alanine, valine, leucine, isoleucine, tyrosine, proline, hydroxyproline, serine, threonine, cystine, cysteine, aspartic acid, glutamic acid, arginine, lysine, histidine, carnitine, and ornithine, (c) a dicarboxylic acid of 3 to 22 carbon atoms, or (d) a carboxylic acid selected from one or more of the group consisting of glycolic acid, pyruvic acid, lactic acid, enolpyruvic acid, lipoic acid, pantothenic acid, acetoacetic acid, p-aminobenzoic acid, betahydroxybutyric acid, orotic acid, and creatine, provided that at least one of said substituents R1, R2 and R3 is not hydrogen, and further provided that if any of said substituents R1, R2 and R3 is hydrogen and if said remaining substituents are acyl radicals of a straight chain fatty acid, then said straight chain fatty acid has 8 to 22 carbon atoms, or a pharmaceutically acceptable salt thereof.
Dwg.0/11
ABEQ US 5583117 A UPAB: 19970122
Delivery of exogenous **uridine** to the tissue of animal, comprises administering to animal, an acyl deriv. of **uridine** of (I) or the pharmaceutically acceptable salt thereof. In (I), R is H or an acyl group derived from carboxylic acid selected from acetic acid, glycolic acid, pyruvic acid, lactic acid, enolpyruvic acid, aminoacid, fatty acid, lipoic acid, pantothenic acid, succinic acid, fumaric acid, adipic acid, acetoacetic acid, p-aminobenzoic acid, beta-hydroxybutyric acid, orotic acid, and creatine; provided that at least one R is not H.
Dwg.0/10

L98 ANSWER 8 OF 11 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD
AN 1987-295462 [42] WPIDS
DNC C1987-125568
TI Depressants acting on **CNS** - contain N3-benzyl-**uridine** derivs. as active component.
DC B03
PA (NISR) NISSUI SEIYAKU KK
CYC 1
PI JP 62207218 A 19870911 (198742)* 6p
JP 06021073 B2 19940323 (199415) 7p A61K031-70
ADT JP 62207218 A JP 1986-49250 19860306; JP 06021073 B2 JP 1986-49250 19860306
FDT JP 06021073 B2 Based on JP 62207218
PRAI JP 1986-49250 19860306
IC A61K031-70; C07H019-06
ICM A61K031-70
ICS C07H019-06
ICA C07H019-067
AB JP 62207218 A UPAB: 19930922
CNS depressants contg. a **uridine** deriv. of formula (I) as active component, (wherein R1 is H, halogen atom or lower alkyl; R2 is H or lower alkyl; R3, R4 and R5 are H or lower alkyl) is novel.
I may be prepd. from N-unsubstd. **uridine** derivs. of formula (II) on reaction with a benzyl halide deriv. of formula (III) (wherein

Sugar means the sugar part of I; X is halogen atom). The reaction is carried out in an inert solvent, e.g. DMFA, acetone, in presence of a base, e.g. K2CO3, Na2CO3, at a temp. of 4-150 deg.C for period of 1-50 hrs.

USE/ADVANTAGE - I can be used as psychotropic agents, antianxiety agents, anti-epileptics or muscular relaxants in a form of oral prepns, or parenteral prepns, injection for lateral ventricle, at a dose of 25-50 mg/60 kg for an adult in oral admin. Also applicable to continuous sleep disorder or agrypnia at a dose of 50-60 mg/60 kg.

/3

FS CPI
FA AB; DCN
MC CPI: B04-B03A; **B12-C05**; **B12-C10**; **B12-D04**; **B12-E02**

L98 ANSWER 9 OF 11 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD
AN 1987-017819 [03] WPIDS
DNC C1987-007290
TI New phosphono methyl **uridine** cpds. - useful as agrochemicals and antitumour, antiviral and **psychotropic** agents.
DC B03 C01
PA (SUNR) SUNTORY LTD
CYC 1
PI JP 61275291 A 19861205 (198703)* 14p
ADT JP 61275291 A JP 1985-114750 19850528
PRAI JP 1985-114750 19850528
IC A01N057-24; A61K031-70; C07H019-06
AB JP 61275291 A UPAB: 19930922
Phosphonomethyluridines of formula (I) and their salts are new. In (I), R1 is H or aralkyl; R2 and R3 are each H or lower alkyl.

Prodn. of (I) comprises (1) reaction of an aldehyde (II) or its equiv. (III) with a dialkyl phosphite (IV) in the presence of a base and (2) conversion of the hydroxy gp. at position 1' of the sugar moiety into the corresp. activated thio ester, followed by reductive deprotection and opt. dealkylation. R4 is a protecting gp. or aralkyl; R5 is a protecting gp.; R5 and R8 each is lower alkyl.

USE - (I) are useful as antagonists against nucleic acid biosynthesis and may be used as antitumour agents, antiviral agents, psychotropic agents or agrochemicals (e.g. insecticides).

0/0

FS CPI
FA AB; DCN
MC CPI: B04-B03B; B12-A06; **B12-C10**; B12-G01; B12-G07; B12-N02; C04-B03B; C12-A06; **C12-C10**; C12-G01; C12-G07; C12-N02

L98 ANSWER 10 OF 11 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD
AN 1986-101874 [16] WPIDS
DNC C1986-043593
TI Use of **uridine** in preventing brain neuron activity **decay** - e.g. in senility, cerebral hypoglycaemia, hypoglycaemia and ischaemia.
DC B03
IN DELUCA, G; DISTAZIO, G; MATERAZZI, M; POLITI, V
PA (POLI-N) POLIFARMA SPA
CYC 4
PI EP 178267 A 19860416 (198616)* EN 10p
R: DE FR GB
IT 1178044 B 19870903 (199035)
PRAI IT 1984-48983 19841009
REP 6.Jnl.Ref; A3...8838; FR 4260; No-SR.Pub
IC A61K031-70
AB EP 178267 A UPAB: 19930922
The use is claimed of **uridine** (I) for prodn. of a medication for counteracting decay in neurone functional activity in brain pathologies, partic. for treatment of cerebral hypoglycemia, hypoxemia, ischemia or senility. Compsn. comprising (I) are also claimed.

Admin. may be p.o. or parenterally. Pref. oral dose is 1-5g/day.

The effect of (I) on somatostatin levels in rat cerebral cortex and hippocampus was investigated following insulin-induced hypoglycemia, since somatostatin levels are reduced in hypoglycemia as well as in the brains of elderly persons. In rats given 15 mg/kg (I), somatostatin levels were normalised in the hippocampus and were even increased in the motor cortex, c.f. a 50% reduction in absence of (I).

0/0

FS CPI
FA AB
MC CPI: B04-B03A; **B12-C10**; B12-G04

L98 ANSWER 11 OF 11 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD
AN 1985-213740 [35] WPIDS
DNC C1985-092992
TI **Sleep** accelerator - contg. **uridine** as active component.
DC B03
PA (KOMO-I) KOMODA Y
CYC 1
PI JP 60136515 A 19850720 (198535)* 3p
ADT JP 60136515 A JP 1983-243483 19831223
PRAI JP 1983-243483 19831223
IC A61K031-70; C07H019-06
AB JP 60136515 A UPAB: 19930925
This sleep accelerator is discovered from low molecular fraction of extract from vigilant rat brain stem, and the active ingredient to accelerate both slow wave sleep and paradoxical sleep is identified as **uridine**. **Uridine** is obtd. by hydrolysis of ribonucleic acid, dephosphorisation of uridylic acid, chemical synthesis and dehydration of 2,6-diketopyrimidine N-2,3,5-tribenzoyl ribofuranoside.

Common dose is 1-100 ng/kg, as **uridine** is water soluble, may be prepd. as injections, and also tablets, granules, and parenteral drugs. Assay of sleep accelerating effect by **uridine** is proceeded by using BDF1 mice (female) and Sprague - Dawley rat (male), **uridine** saline soln. is administered into abdominal (mice), and diacele by cannula (rat), both slow wase sleep and paradoxical sleep are significantly accelerated.

0/0

FS CPI
FA AB
MC CPI: B04-B03; **B12-C07**; **B12-C08**